“CLINICAL STUDY OF DERMOSCOPIC FINDINGS IN NON-CICATRICIAL & CICATRICIAL ALOPECIA”

Dissertation Submitted in

Partial fulfillment of the University regulations for

MD DEGREE IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

(BRANCH XX)

MADRAS MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, INDIA.

APRIL 2015

CERTIFICATE

Certified that this dissertation titled "**CLINICAL STUDY OF DERMOSCOPIC FINDINGS IN NON-CICATRICIAL & CICATRICIAL ALOPECIA"** is a bonafide work done by **Dr. VIVEK SHAH,** Post-graduate student of the Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2012 – 2015. This work has not previously formed the basis for the award of any degree.

Prof. Dr. K. MANOHARAN M.D., D.D.,
Professor and Head
Department of Dermatology
Madras Medical College
Chennai - 3

Prof Dr. R.VIMALA M.D.,
Dean
Madras Medical College
Chennai – 3

DECLARATION

The dissertation entitled **"CLINICAL STUDY OF DERMOSCOPIC FINDINGS IN NON-CICATRICIAL & CICATRICIAL ALOPECIA"** is a bonafide work done by **Dr. VIVEK SHAH** at Department of Dermatology, Venereology and Leprosy, Madras Medical College, Chennai – 3, during the academic year 2012 – 2015 under the guidance of **Prof. Dr.V.SAMPATH M.D.,** Professor, Department of Dermatology, Madras Medical College, Chennai -3.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of **M.D Degree in Dermatology Venereology and Leprosy (BRANCH – XX)**

Prof. Dr. V. SAMPATH, M.D.,
Professor,
Department of Dermatology,
Madras Medical College,
Chennai-03

DECLARATION

I, **Dr. VIVEK SHAH** solemnly declare that this dissertation titled "**CLINICAL STUDY OF DERMOSCOPIC FINDINGS IN NON-CICATRICIAL & CICATRICIAL ALOPECIA**" is a bonafide work done by me at Madras Medical College during 2012-2015 under the guidance and supervision of **Prof. Dr. K.MANOHARAN, M.D.,D.D.,** Professor and Head of Department of Dermatology, Madras Medical College, Chennai-600003.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai towards partial fulfillment of the rules and regulations for the award of **M.D Degree in Dermatology Venereology and Leprosy (BRANCH – XX)**

PLACE :

DATE :

(DR. VIVEK SHAH)

SPECIAL ACKNOWLEDGEMENT

My sincere thanks to **Prof. Dr. R.Vimala M.D.,** Dean, Madras Medical College for allowing me to do this dissertation and utilize the Institutional facilities.

ACKNOWLEDGEMENT

I am gratefully indebted to Professor and Head of the Department of Dermatology, **Prof. Dr. K. MANOHARAN M.D., D.D.,** for his invaluable advice, guidance and encouragement throughout the study.

I would like to express my sincere and heartfelt gratitude to **Prof. Dr. V.SUDHA M.D., D.V., D.D.,** Director and Professor, Institute of Venereology, for her kindness and support throughout the study.

I sincerely thank **Prof. Dr. C. JANAKI M.D., D.D.,** Professor of Dermatology for her priceless support.

I thank my Professor and Head of the Department of Occupational and Contact Dermatitis, **Prof. Dr. S. NIRMALA M.D.,** for her help and support.

I express my sincere gratitude to my guide **Prof. Dr. V. SAMPATH M.D.,** Professor of Dermatology for his guidance and support.

I also thank **Prof. Dr. R. PRIYAVATHANI M.D., D.D., DNB.,** Professor of Dermatology for her advice and encouragement.

I am grateful to **Prof. Dr. U.R. DHANALAKSHMI M.D., D.D.,** Additional Professor, Department of Dermatology for her invaluable guidance and help.

I am grateful to **Prof. Dr. S. KALAIVANI M.D., D.V.,** Additional Professor, Institute of Venereology for her guidance and help.

I would also like to thank **Prof. Dr. K. VENKATESWARAN M.D., D.V.,** former Additional Professor, Institute of Venereology for his timely help.

I wish to thank **Prof. Dr.. R. ARUNADEVI M.D., D.D.,** former Professor of Dermatology for their support and motivation.

I humbly thank my Co-Guide **Dr. N. SARAVANAN M.D. D.V.L.,** for his valuable guidance throughout my work.

I extend my gratitude to **Dr. G.K. THARINI M.D.(Derm), Dr. R. MADHU M.D.(Derm), D.C.H., Dr. V. N. S. AHAMED SHARIFF M.D. D.V.L., Dr. SAMUEL JEYARAJ DANIEL M.D. D.V.L., Dr. K. UMA MAHESHWARI, M.D. D.V.L., Dr. VIJAYALAKHSMI, M.D. D.V.L. and Dr. NITHYA GAYATHRI DEVI, M.D. D.V.L.** Assistant professors, Department of Dermatology for their kind support and encouragement.

I also thank my Assistant Professors **Dr. P. MOHAN M.D., D.V., Dr. P. PRABHAKAR M.D.D.V.L., Dr. C. VIDHYA M.D.DVL., Dr. DEEPA M.D.DVL, Dr. S. VENKATESAN D.V., DNB (D.V.L.),**

Dr. V. GOMATHY M.D. D.V.L. and **Dr. R. MANIPRIYA M.D. D.V.L., D.C.H.** of Institute of Venereology for their able guidance.

I express my thanks to my former assistant professors, **Dr. C. VIJAYABHASKAR M.D., D.CH., Dr. J. MANJULA M.D., DNB., & DR. S. MADHAVI M.D. D.V.L.,** Department of Dermatology, for their support and help.

I wish to thank **Dr. R. SOWMIYA, M.D.D.V.L., Dr. V. SENTHILKUMAR DNB., D.STD, Dr. R. SUBHA, M.D.DVL., Dr. N.S. JAYANTHI, M.D.D.V.L., & Dr. S. SANGEETHA, D.D.V.L.,** former Assistant Professors, Institute of Venereology for their constant guidance.

I am thankful to my colleagues **Dr. SNEHAL TAPADIYA, Dr. KHUSHBOO MINNI** and **Dr. MONIL NAGAD** for their support throughout the study.

I am also grateful to all paramedical staffs for rendering timely help to complete my study.

I am also extremely thankful to my family for their motivation and encouragement.

Last but not the least I am profoundly grateful to all patients for their co-operation and participation in this study.

CONTENTS

ABSTRACT

INTRODUCTION: The diagnosis of alopecia is based on clinical grounds. However, sometimes clinical evaluation may not provide a clear diagnosis and in such cases, scalp biopsy and histopathological examination may provide us with the diagnosis. Dermoscopy is a non-invasive tool using which we can make the diagnosis in clinically doubtful cases and a scalp biopsy can be avoided.

AIMS AND OBJECTIVES: To study the dermoscopic findings in non-cicatricial & cicatricial alopecias and to study the correlation between dermoscopic findings & histopathological examination, in cases of alopecia with doubtful diagnosis.

METHODOLOGY: 82 patients with alopecia over a one year period were included in the study. Detailed clinical examination and scalp dermoscopy were done in all patients. Histopathological examination was done in patients with doubtful diagnosis. Scalp scraping and hair root examination was done in suspected cases of tinea capitis.

RESULTS: The dermoscopic findings in patients with alopecia in our study were consistent with those mentioned in literature with some differences. This includes more common occurrence of black dots than yellow dots in alopecia areata, equal specificity of coiled hair and cockscrew

hair when compared with comma hair for diagnosing tinea capitis and occurrence of Wickham's striae in lichen planopilaris. Lipid globules for diagnosing nevus sebaceous and stellate follicular scars for diagnosing aplasia cutis have also been reported in our study.

CONCLUSION: Dermoscopy is non-invasive tool which can help us in achieving an etiological diagnosis in patients with alopecia without resorting to a scalp biopsy.

INTRODUCTION

Alopecia refers to loss of hair from any part of the body. Scalp is the most common and most disfiguring site of alopecia for patients. Alopecia can be broadly classified into non-scarring and scarring alopecia.

The diagnosis of alopecia is based on clinical grounds. However, sometimes clinical evaluation may not provide a clear diagnosis and in such cases, scalp biopsy and histopathological examination may provide us with the diagnosis. Dermoscopy is a non-invasive tool using which we can make the diagnosis in clinically doubtful cases and a scalp biopsy can be avoided.

Dermoscopy can be used to analyse various features of the scalp and hair. Dermoscopic findings have a histopathological correlation and help in better understanding of the underlying pathologic process. Dermoscopic findings also have a prognostic significance.

This study has been done to explore the role of dermoscopy in diagnosing various alopecia in Indian skin as most of the previously conducted studies evaluating use of dermoscopy for diagnosing alopecia have been done on fair skin. An attempt has also been made to correlate dermoscopic findings with histopathological findings in certain cases.

Review Of Literature

REVIEW OF LITERATURE

Alopecia can be broadly classified into scarring and non-scarring alopecia.

Non-scarring alopecia include:[1]

- Congenital and hereditary alopecia
    - Congenital universal atrichia
    - Atrichia with papules
    - Hereditary vitamin D resistant rickets
    - Ectodermal dysplasia
    - Trichophalangeal syndrome 1
- Alopecia due to hair cycle defect
    - Telogen hair loss- Telogen effluvium, Androgenetic alopecia, Alopecia areata, postpartum alopecia, neonatal alopecia, hypothyroidism, drugs.
    - Anagen hair loss- Anagen effluvium, loose anagen hair syndrome
    - Short anagen syndrome
- Alopecia due to hair shaft defects
    - Hair fractures
    - Hair nodes

    - Hair narrowings
    - Hair twists and curls
    - Hair bands
    - Unruly hair
- Others
    - Alopecia subtotalis/ totalis
    - Alopecia universalis

Scarring alopecia include:[1]

- Developmental/ Hereditary:
    - Aplasia cutis
    - Focal dermal hypoplasia
    - Dystrophic epidermolysis bullosa
    - Incontinentia pigmenti
    - Conradi syndrome
    - Keratosis pilaris atrophicans faciei
    - Keratosis follicularis spinulosa decalvans
    - Darier's disease
    - Porokeratosis of Mibelli
    - Epidermal Naevus
    - Lamellar icthyosis
    - Marie-Unna syndrome

    - Follicular hamartoma syndrome
    - Polyostotic fibrous dysplasia
    - Hallermann-Streiff syndrome
    - Rapp- Hodgkin syndrome
- Traumatic
    - Mechanical
    - Thermal
    - Electrical
    - Tractional/ trichotillomania (persistent)
    - Factitious injury
- Neoplastic
    - Basal cell carcinoma
    - Squamous cell carcinoma
    - Syringolymphoid hypoplasia
    - Cutaneous angiosarcoma
    - Lymphomas
    - Metastasis
- Follicular inflammation with pustules
    - Tinea capitis
    - Bacterial folliculitis
    - Folliculitis decalvans

    - Dissecting cellulitis of scalp
    - Acne keloidalis nuchae
    - Tufted folliculitis
    - Hidradenitis suppurativa
- Follicular inflammation without pustules
    - Pseudopelade of Brocq
    - Follicular lichen planus
    - Graham Little syndrome
    - Discoid lupus erythematosus
    - Lichen sclerosus et atrophicans
    - Follicular mucinosis
- Dermal inflammation leading to secondary follicular damage
    - Cicatricial pemphigoid
    - Morphea
    - Facial hemiatrophy
    - Dermatomyositis
    - Necrobiosis lipoidica
    - Sarcoidosis
    - Temporal arteritis
    - Pyoderma gangrenosum
    - Syphilis

- Tuberculosis
- Leishmaniasis
- Myiasis
- Herpes zoster
- Ecthyma and deep secondary pyodermas

Variety of investigations can be carried out to confirm the cause of alopecia. These include: [1]

- Clinical tests:
  - Hair pull tests
  - Daily hair counts
  - Hair feathering
  - Wood's lamp examination
  - Hair weight examination
  - Trichometry
  - Trichotillometry
  - Hair growth window
- Microscopic examination of hair
  - Trichogram/ hair pluck test
  - Photographic trichogram
  - Trichoscopy
  - Light microscopy of hair

    - Polarizing microscopy of hair
    - Confocal microscopy of hair
    - Electron microscopy of hair
- Scalp biopsy
- X-ray diffraction studies

TRICHOSCOPY

Trichoscopy is the dermoscopic examination of the scalp. Trichoscopy can be performed using handheld or digital dermoscopes Videodermoscopes). Handheld dermoscopy may be performed using dermoscopes which require linkage fluids or those using polarized light (with or without contact lens). Videodermoscopy is expensive and time consuming but has advantages of easier photography and higher magnification.

Keratotic structures are better viewed in non-polarized light whereas blood vessels are seen better seen with polarized dermoscopy.[2]

Hand held dermoscopes provide a magnication of 10 times whereas videodermoscopes have a magnification power of 20-to 100- fold. Lower magnification aids in the rapid scanning of scalp whereas higher magnification is essential for viewing fine details.

Non-polarized dermoscopes may be used without using linkage fluid for visualisation of perifollicular scaling (Dry trichoscopy).[3] Linkage fluids

which can be used for trichoscopy include aqueous solutions like distilled water/ saline, alcoholic skin disinfectants or gels.

Dermoscopy of scalp and hair can give valuable information about hair shaft abnormalities, hair follicle opening alterations and dots, perifollicular and interfollicular skin and scalp vascular changes.

I. Hair shaft abnormalities and their causes include: [5]

- Fractured hairs
    - Trichoptilosis
    - Trichoschisis/ Trichoclasis
    - Irregular fractures caused by mechanical force
    - Golf tee hairs (trichorrhexis invaginata)
- Narrowings
    - Monilethrix
    - Monilethrix-like congenital hypotrichosis
    - Pohl- Pinkus constriction
    - Pseudomonilethrix
    - Tapered hair
    - Exclamation mark hair
- Node-like appearance
    - Trichonodosis
    - Trichorrhexis nodosa

    - Bamboo hair (Trichorrhexis invaginata)
    - Hair casts
- Curls and twists
    - Pigtail hair
    - Coiled hair
    - Comma hair
    - Cockscrew hair
    - Zigzag hairs
    - Pili torti
    - Wooly hair
- Bands
    - Interrupted medulla
    - Continuous medulla
    - Pili annulati
    - Interrupted (Morse code-like) hairs
- Short hairs
    - Upright regrowing hair
    - Vellus hairs
    - Dark lines
    - Tulip hairs
    - Block hairs

- i-Hairs
- Broom hairs
- Flame hairs

II. Hair follicle opening changes: [5]

- Black dots
  - Alopecia areata
  - Tinea capitis
  - Trichotillomania
  - Dissecting cellulitis of scalp
  - Rare causes:
    - Monilethrix
    - Chemotherapy induced alopecia
    - Traction Alopecia
- Yellow dots
  - Alopecia areata
  - Androgenetic alopecia
  - Dissecting cellulitis
  - Discoid lupus erythematosus
- White dots
  - Cicatricial alopecia (lichen planopilaris)
  - Empty hair follicles

III. Perifollicular and interfollicular surface changes: [5]

- Scaling
  - Diffuse
    - White scales-Psoriasis, Discoid lupus erythematosus
    - Yellow scales- Seborrheic dermatitis, Discoid lupus erythematosus
  - Perifollicular
    - White scales- Lichen planopilaris
    - Yellow scales- Folliculitis decalvans
- Colour
  - Brown pigmentation
    - Physiological reticular pattern
    - Peripilar sign (androgenetic alopecia, telogen effluvium)
    - Scattered (discoid lupus erythematosus)
  - White areas (cicatricial alopecia)
  - Strawberry ice cream like (early cicatricial alopecia)
  - Yellow areas (Folliculitis decalvans, dissecting cellulitis)
  - Red (Inflammation, extravasation, vascular abnormalities)

IV. Vascular changes: [5]

- Comma vessels, dotted vessels, glomerular vessels - Inflammatory conditions of scalp like psoriasis and seborrheic dermatitis.
- Hair pin vessels and concentric perifollicular vessels- Cicatricial alopecia especially lichen planopilaris and folliculitis decalvans
- Arborizing vessels and serpentine vessels- Discoid lupus erythematosus

A complete analysis of all these findings and its correlation with clinical features may help us in clinching a diagnosis. The first dermoscopic examination may not give a conclusive diagnosis in some cases. In such a scenario, it is better to perform a repeat dermoscopic examination after an interval of time. Comparison between successive dermoscopic images and images from different scalp areas is often useful in establishing a diagnosis. Scalp biopsy should be the last resort for confirming the diagnosis.

Dermoscopic findings have prognostic significance and comparison between dermoscopic images is a non-invasive method of assessing response to therapy.

SCALP BIOPSY

Usually, a 4 mm punch biopsy is performed over the scalp skin in order to obtain enough hairs to analyse the pathological process. The biopsy specimen can be subjected to vertical and horizontal sectioning. In vertical sectioning, the cylinder of tissue is bisected longitudinally whereas in horizontal sectioning, the biopsy sample is “bread-loafed”.

Examination of both the sections gives useful information and examination of both sections have their own advantages and disadvantages. Horizontal sectioning allows viewing of all the follicles at a particular level whereas only 10-15% of the hairs in a sample can be viewed in a single vertical section. Vertical sectioning allows more comfortable visualisation of entire hair follicle anatomy along with the epidermis. However, horizontal sectioning does not allow proper visualisation of hair follicle anatomy.

Horizontal sections have some definite advantages over vertical section. These include:

- Availability of all hair follicles for examination in a single section
- Rapid evaluation of hair density and follicular units
- Correct assessment of follicular size
- Pathologic alterations at different levels of hair follicles can be examined
- Quantitative evaluation is possible

Most dermatologists prefer taking two 4-mm punch specimens. One is sectioned horizontally and the other is sectioned vertically. A part of the vertical sectioned specimen may be sent for direct immunofluorescence in case of suspected lichen planopilaris, discoid lupus erythematosus and in cases of cicatricial alopecia where no specific diagnosis can be established.

In order to differentiate early androgenetic alopecia and telogen effluvium, some dermatologists prefer taking two specimens. One specimen is taken from the frontal region and one from the occipital region. In androgenetic alopecia, it is expected that the pathologic process will involve the hair follicles in the frontal area and spare the occipital area. However, in telogen effluvium all areas of the scalp are equally involved.

In order to identify the etiological diagnosis in cicatricial alopecia, the biopsy specimen should be taken from the edge of the lesion where there is new hair loss, evidence of signs of inflammation or a positive hair pull test. In order to assess the possibility of regrowth of hair in a scarred area, the biopsy sample has to be taken from the central “burnt-out” area. [4]

Histopathological examination may not provide a definite diagnosis in a minority of cases. In these patients, a correlation of clinical, dermoscopic and histopathological findings may provide a diagnosis and give clues which may help in deciding the line of management.

On the basis of histopathological features, primary scarring alopecia may be classified as:

- Lymphocytic alopecias:
    - Chronic cutaneous lupus erythematosus
    - Lichen Planopilaris
        - Classic Lichen Planopilaris
        - Graham-Little syndrome
        - Frontal Fibrosing Alopecia
    - Pseudopelade of Brocq
    - Central Centrifugal cicatrical alopecia
    - Alopecia mucinosa
    - Keratosis pilaris spinulosa decalvans
- Neutrophilic
    - Folliculitis decalvans
    - Dissecting cellulitis/ folliculitis (including tufted folliculitis)
- Mixed
    - Acne keloidalis
    - Acne necrotica
    - Erosive pustular dermatosis
- Non-specific or end stage cicatricial alopecia

MICROSCOPIC EXAMINATION OF HAIR

FOR FUNGAL ELEMENTS

- Scalp scraping and hair root examination are the two specimens routinely examined for establishing the diagnosis of tinea capitis.
- These investigations are also extremely useful to differentiate alopecia areata and alopecia areata type of tinea capitis.
- The specimens are mounted in 10% potassium hydroxide solution. Hairs are fragile specimens and leaving them in potassium hydroxide for more than a few minutes or excessive heating may distort the morphology of the arthroconidia. 10% sodium sulphide solution can be used in place of 10% potassium hydroxide solution.
- The slide should be examined immediately after mounting. The slide should be viewed initially in low power (×10) and the presence of fungus should be confirmed in high power (×40).
- The presence of hyphal elements and refractile arthrospores is confirmatory of the tinea capitis.

Hair Shaft Defects

Monilethrix

- It is an autosomal dominant disorder of hair shaft with regular thinning of hair shaft with fracture of the hair shaft at constricted points.
- It is commonly caused due to human hair keratin hHb6 gene. [6]
- Patients have short and fragile hair not requiring haircut since childhood commonly over the occipital and temporal scalp but may also involve eyebrows, eyelashes, axillary, pubic or body hair. [7]
- Trichoscopy shows uniform nodosities with intermittent constrictions at which there is shaft breakage. This is called "Regularly bent ribbon sign". Horny follicular papules appear as big yellow dots with immersion fluid. Perifollicular scaling and keratotic plugs may be seen on dry dermoscopy. [8]

Pseudomonilethrix

- It is irregular square shaped flattening of hair shafts which is considered by some as reaction to excessive use of cosmetics and some as an artifact.[9]
- It is to be differentiated from monilethrix-like hairs which are seen in monelithrix-like congenital hypotrichosis, alopecia areata, primary

cicatricial alopecia and lichen planopilaris. Chemotherapy, immersion fluid or styling gel may lead to monilethrix-like hairs. [8]

Trichorrhexis Invaginata

- Netherton's syndrome is an autosomal recessive disorder with icthyosis linearis circumlexa, atopic diathesis and trichorrhexis invaginata. Trichorrhexis invaginata has invagination of hair shafts within themselves at the keratinization zone. [7]
- Patients have sparse, short, dry, brittle and spiky hair. Trichoscopy shows multiple small nodules occurring at irregular intervals with higher magnification showing distal part invaginating into proximal hair shaft giving "ball in cup" appearance. Ragged proximal end with fractured distal end gives "golf tee" appearance. This can be demonstrated over scalp and eyebrow hair. [10]

Trichorrhexis Nodosa

- Hair shaft splits longitudinally into multiple small fibers which bulge outward leading to a segmental increase in hair diameter, ultimately breaking leaving brush-like ends.[11]
- Patients have dry and brittle hairs which break at different lengths.
- Trichoscopy shows nodular thickenings of hair shaft which are lighter in darker hair shafts. The hair has bends with rounded edges

at the level of the nodule. Broken hair shafts are lighter than remaining hair shafts. Higher magnification reveals numerous small fibers producing appearance of two brushes aligned in apposition or brush like ends in broken hairs.[12]

Pili Torti

- Twisted hairs which show flattened hair shafts at irregular intervals and then rotated 180º around its long axis. (13)
- Dry and brittle hair which break easily are commonly seen over occipital and temporal scalp but may also be seen over eyebrow, eyelashes and axillary hair.
- It can have an early onset (Ronchese/ Classic type) or late onset (Beare type)
- Trichoscopy reveals twisted hair shafts along long axis with lower magnifications showing bending of hair shafts at different angles. [14]

Pili Annulati

- Autosomal dominant disorder characterized by hair shafts with alternating white and dark bands.
- It appears at birth or infancy with clinically demonstrable hair with dark and light bands which are otherwise normal, commonly seen over scalp but may involve axillary, beard or pubic hairs. [15]

- Trichoscopy reveals alternatively arranged light and dark bands with reduction in the number of white bands distally. [14]
- It has to be differentiated trichoscopically from fragmented medulla with a longitudinal white structure covering less than 50% of hair shaft width whereas pili torti covers more than 50% of hair shaft thickness. Pseudo-pili torti with partial twisting of hair without white bands also needs to be differentiated.

Trichothiodystrophy

- It is characterized by sulphur deficient brittle hair associated with photosensitivity, brittle hair, icthyosis, impaired intelligence, decreased fertility, short stature and fertility. (BIDS, IBIDS and PIBIDS)
- Fragile, unruly and brittle hair are seen over scalp hair, eyebrows and eyelashes.[16]
- Light microscopy shows trichoschisis (irregular undulating contour with clean transverse fractures). Polarized light shows characteristic "tiger tail bending".[17]
- Trichoscopy shows non-homogenous structure like grains of sands, wavy contour, trichoschisis and trichoclasis.[14]

Other congenital or genetically mediated alopecias

Ectodermal dysplasia

- Abnormalities of two or more tissues of ectodermal origin including hair, teeth, nails, sweat glands, and other tissues.[18]
- The common types of ectodermal dysplasia include:
    - Anhidrotic ectodermal dysplasia/ Hypohydrotic ectodermal dysplasia/ Christ-Siemens-Touraine syndrome
    - Hidrotic ectodermal dysplasia/ Clouston syndrome
    - Ankyloblepharon Filiforme Adnatum- Ectodermal dysplasia-Cleft Palate syndrome (Hay-Wells syndrome and Rapp-Hodgkin syndrome)
    - Ectrodactyly- Ectodermal dysplasia- Cleft lip/ cleft palate syndrome
    - Tooth and Nail syndrome (Witkop syndrome)
- These patients have sparse, thin, curly, dry and brittle hair over scalp. Eyebrows and eyelashes may be absent or sparse.[19]
- Trichoscopy shows increased follicular units with single hair, hair shaft heterogeneity, pili torti, trichoschisis, pili canaliculi, trichorrhexis nodosa, monilethrix-like hairs or trichothiodystrophy like hairs.[20]

Nevus sebaceous

- It is an epidermal hamartoma composed of sebaceous glands usually seen over the head and neck region.
- These are usually sporadic and may indicate lethal mutations rescued by mosaicism. Mutations of PTCH and Gli-1 have been implicated in the pathogenesis of nevus sebaceous.
- It usually presents as an orange, yellow, pink or tan hairless plaque at birth with a smooth or velvety surface. The lesions usually remain unchanged until puberty and then become elevated and verrucous.
- Most of the nevi occur over the head and neck region.
- These are prone to develop variety of benign or malignant tumors. The benign tumors include syringocystadenoma papilliferum, trichoblastoma, infundibuloma, nodular hidradenoma, syringoma, apocrine cystadenoma, pilomatricoma, trichilemmoma and trichoadenoma. The malignant tumors include keratoacanthoma, proliferating trichilemmal cyst, sebaceous, basal cell, apocrine, eccrine, squamous carcinomas and malignant melanomas.
- Histopathological examination shows cords of poorly differentiated epithelial cells which represent primordial pilosebaceous units. After puberty, mature sebaceous glands with characteristic hyperplasia of overlying epidermis are seen. Hair follicles are inconspicuous but ectopic apocrine glands may be seen deep in the dermis.

- Excision of the lesion is the best treatment option as there is risk of future neoplastic transformation. Other options include removal by dermabrasion or carbon dioxide laser. However, the last 2 options may be followed by future recurrence.

Aplasia cutis

- Aplasia cutis refers to failure of skin development whereas congenital absence of skin also includes those conditions where skin development has occurred but was subsequently lost.
- Histological variations include absent epidermis and dermis, presence of dermis with absence of appendages and elastic tissue, absence of subcutaneous tissue, associated dural or skull defect. During re-epithelisation, epidermis is usually flat with absent appendages. Hypertrophic scarring may occur.
- Freiden classified aplasia cutis into various types:

Type 1 : Non-syndromic aplasia cutis congenital of the scalp

Type 2 : Congenital absence of skin on the scalp with limb reduction defects/ Adams-Oliver syndrome

Type 3 : Congenital absence of skin on the scalp with epidermal nevi

Type 4 : Congenital absence of skin overlying developmental malformations

Type 5 : Congenital absence of skin associated with fetus papyraceus

Type 6 : Congenital absence of skin with epidermolysis bullosa/ Bart's syndrome

Type 7 : Isolated congenital absence of skin localized to the extremities

Type 8a : Congenital absence of skin caused by specific teratogens

Type 8b : Congenital absence of skin following intrauterine infections

Type 9 : Congenital absence of skin as a feature of malformation syndromes

- Aplasia cutis is a congenital scarring alopecia and differentiation of this entity from other congenital scarring alopecia is essential as aplasia cutis has frequent systemic associations. A detailed systemic examination with special emphasis on central nervous system examination should be performed in all cases of aplasia cutis over the scalp.

- Most of the lesions heal spontaneously. The other lesions may be subjected to excision of abnormal skin margins with primary closure, grafting or flap rotation (for large scalp defects) or tissue expansion techniques.

NON-CICATRICIAL ALOPECIA

Alopecia areata

- It is a non-scarring alopecia with complex etiology involving genetic, environmental and immunologic factors.
- Ikeda classified alopecia areata into common type, atopic type, autoimmune type and pre-hypertensive type.[1]
- Common type (81%) accounts for maximum cases and is not associated with other disorders. Middle aged patients are commonly affected .The chance of developing alopecia totalis is 5-15% with individual patches usually lasting for less than six months.
- Atopic type (10%) starts during childhood with individual patches lasting for more than on one year. Reticular and ophiasis pattern are common with the chance of alopecia totalis being 30-75%.
- Autoimmune type accounts for 5% of the cases. It is associated with diabetes mellitus, peptic ulcer disorders and other autoimmune conditions. Chance of alopecia totalis is high in this type accounting for 10-50% cases.
- Prehypertensive (4%) has fast progression with increased chance of alopecia totalis in about 40% cases. Reticular pattern is common in this type.

- Clinically, alopecia areata may be classified into:
    - Based on extent: Patchy alopecia, alopecia totalis, alopecia universalis
    - Based on pattern: Reticular, ophiasis, sisaipho
    - New variants: Acute and diffuse total alopecia
    - Unusual patterns: Perinevoid alopecia, Linear alopecia areata
- Patchy alopecia areata is most common clinically and is characterized by round to oval, smooth patches of hair loss which sometimes coalesce with each other without any evidence of scarring.[21]
- The trichoscopic findings for alopecia areata include yellow dots(regularly distributed), black dots, broken hair, exclamation mark hair, tapering hair and regrowing upright hair.[22]
- Active alopecia areata shows black dots, broken hair, microexclamation mark hair, monilethrix-like hairs and trichorrhexis nodosa.Inactive alopecia areata shows yellow dots and vellus hair. Hair growth is indicated by upright regrowing hair, pigtail hair and vellus hair.[23]
- Yellow dots are regularly arranged in groups of two to three with comparatively lesser number in frontal scalp differentiating it from those seen in androgenetic alopecia.

- Broken hairs have an uniform length differentiating it from trichotillomania. The broken hair may show a Pohl-Pinkus constriction or monelithrix-like hair before it actually breaks.
- Dermoscopic coudablity/ zig-zag hairs is another indicator for hair shaft weakness.
- Microexclamation mark hair are hairs 1-2 mm long which are thin at proximal aspect and thick at distal aspect. Normal exclamation mark hairs, which are visible to the naked eye, are approximately 1 cm, whereas trichoscopy allows visualization of exclamation mark hair that are 1-2 mm long.[24] Microexclamation mark hairs may be seen in trichotillomania but lack the hypopigmented proximal end and distal pointed end seen in Alopecia Areata. Tapered hair are large microexclamation mark hair seen in Alopecia Areata.
- Tulip hairs resemble microexclamation mark hair but the proximal end is mildly thinned with light colour with a dark distal end.
- Histopathological examination shows:
    - Acute stage: Peribulbar and intrabulbar lymphocytic inflammatory infiltrate around anagen hair follicles giving "swarm of bees" appearance.
    - Subacute stage: Increased catagen or telogen hair are seen

- Chronic stage: Follicular miniaturization with ratio of terminal: vellus hair reduced from 7:1 to 1:1.

- The treatment of alopecia areata is sometimes complicated by the fact that lot of patients have spontaneous resolution and thus, it is difficult to assess the therapeutic efficacy of different drugs.
- The topical treatments include:
  - Immunosuppressants like topical corticosteroids
  - Contact sensitizers like diphencyprone and squaric acid dibutyl ester
  - Irritants like salicylic acid, anthralin, tretinoin, canthradin, sulphur and oil of Cade
  - Topical photochemotherapy
- Intralesional steroids are used especially for patchy alopecia areata.
- Systemic therapies include:
  - Immunosupressants like corticosteroids, cyclosporine, mycophenolate mofetil
  - Immunomodulators like alefacept, sulphasalazine and intravenous immunoglobulin
- Other methods for treatment include whole body photochemotherapy with UV-A, cryotherapy, excimer laser, pulsed infrared diode laser, hypnotherapy, tattooing and use of wigs or hair pieces.[1]

Androgenetic Alopecia

- Androgenetic Alopecia is due to androgen sensitivity of hair follicles in genetically predisposed individuals. It affects 80% of Caucasian men and more than 42% of Caucasian women.[25]
- Male pattern hair loss follows the Hamilton's grading. This includes the following:

Grade I	-	Prepubertal smooth outline of frontal scalp
Grade II	-	Inverted V-shaped frontal recession at puberty
Grade III	-	Recession of bitemporal hairline in early adulthood or late twenties
Grade IV	-	Deep frontotemporal recession, some midfrontal recession with some vertical thinning in older ubjects
Grade V	-	Further fronto-temporal recession and marked vertical thinning
Grade VI	-	Further progression with tendency towards Confluence
Grade VII	-	Enlargement of both areas separated only by a fringe of hair

Grade VIII - Confluence of both bald the areas with only a fringe of occipital hair persisting which may be lost later[1]

Female pattern hair loss may be of 3 types:

- Hamilton's type: Fronto-parietal type resembling male pattern hair loss
- Olsen's Christmas tree pattern: Widening of parting line resembling a Christmas tree pattern
- Ludwig type: Progressive centrifugal thinning of hair over frontal region. This may be progressively graded from Grade I to Grade III [1]

- The trichoscopic findings in patients of androgenetic alopecia include hair shaft diameter variation, thin hairs, vellus hairs, single hair pilosebaceous units, yellow dots, perifollicular discolouration, wavy hair & honeycombed pigmentation.[26]
- Hair shaft heterogeneity means presence of hairs of different thickness like vellus, thin, intermediate & thick hairs.
- The number of vellus hairs is increased in androgenetic alopecia (average 20.9%) from a normal percentage of upto 10 %.[27]These are hypopigmented, non-medullated hairs less than 30 µm thick and less than 2-3 mm long. Vellus hair should be differentiated from newly regrowing hair which are upright, pigmented and have a pointed end.

- Peripilar sign or perifollicular brown discolouration which occurs due to perifollicular lymphocytic infiltrate. This may be seen in normal individuals and in telogen effluvium.[28]
- The features of androgenetic alopecias are more marked in frontal than occipital area in both men and women. The criteria for female androgenetic alopecia include three major and minor criteria.

(A) Major criteria include:

(1) More than 4 yellow dots in 4 images in frontal area,

(2) Lower average hair thickness in the frontal area than in occipital area

(3) More than 10% of thin hairs (<0.03 mm) in frontal area.

(B) Minor criteria include increased frontal to occipital ratio of:

(1) Single hair pilosebaceous units

(2) Vellus hairs

(3) Perifollicular discolouration

Fulfilment of two major or one major and two minor criteria allows diagnosis of female pattern hair loss with 98% specificity.[27]

- Histopathological examination shows reduced ratio of terminal:vellus hair to 3:1, increased telogen:anagen ratio, minimal perifollicular infiltrate and increased follicular steleae. Miniaturization of hair is the hallmark which is depicted by random variation in hair follicle caiber.[29]
- Treatment of androgenetic alopecia includes topical and systemic drugs, surgery and supportive therapy.
- Therapeutic options in males include topical therapies like 2-5% minoxidil, tretinoin 0.025%, azelaic acid 5% and ketoconazole 2%. Systemic agents like antiandrogens like finasteride and dutasteride can also be used for treating male androgenetic alopecia. Surgical therapeutic modalities include scalp reduction, hair transplantation-punch graft, follicular unit transplantation and follicular unit grafting. In cases with extensive androgenetic alopecia, supportive therapies like wigs and hair pieces can be used.[1]
- Treatment options for females is similar to male androgenetic alopecia. Systemic antiandrogens like cyproterone acetate, spironolactone and flutamide can also be used in female androgenetic alopecia patients.[1]

Telogen effluvium

- It is characterized by abrupt shedding of telogen hairs commonly triggered by internal or external factors which cause a large number of hair to enter the telogen phase together. This starts 3-4 months after the insult.
- Precipitating factors include
    - Febrile illness
    - Psychological trauma
    - Pregnancy
    - Surgery
    - Thyroid disorders
    - Crash diet and iron deficiency
    - Withdrawal of estrogen- containing medications
    - Drugs (beta-blockers, anti-coagulants, propylthiouracil, carbamazepine, vaccines, retinoids)
    - UV exposure[30]
- Chronic telogen effluvium is characterized by a diffuse loss of telogen hairs persisting for more than 6-8 months. Hair loss with thinning and bitemporal recession may be present mimicking androgenetic alopecia.
- Trichoscopic findings are non-specific and include predominance of hair follicle with single hair, upright regrowing hairs, peripilar brown

discoloration and yellow dots indicating empty follicles. It is diagnosis of exclusion.

- It is to be differentiated from androgenetic alopecia where peripilar discoloration, vellus hairs and hair shaft diameter heterogeneity is more common. The abnormalities are more in the frontal scalp in androgenetic alopecia whereas it is uniform over the scalp in telogen effluvium.[31]
- Histopathology shows normal number of hair follicles with increased number of telogen hair. Telogen hair are identified histologically by trichilemmal keratinization of proximal part of hair shaft. No hair shaft miniaturization is seen.[29]
- Telogen effluvium has no specific therapy. Correction of the causative insult usually ensures hair growth within 6 months. This may however be incomplete. Micronutrient supplementation is a treatment option in case there is deficiency of a specific nutrient for example iron supplementation in iron deficiency.[1]

<u>Anagen effluvium</u>

- It is seen when there is diffuse hair loss from the hair follicles in anagen growth phase due to rapid suppression of mitotic activity by cytotoxic drug or other toxic factors.[1]

- Commonly seen with chemotherapy but it may also be seen with noncytotoxic drugs like acitretin, radiation therapy, toxins and systemic disorders.
- Trichoscopy of anagen effluvium shows black dots, monilethrix like hairs, thinning (Pohl-Pinkus constriction), shaft breakage at sites of constriction, pig tail hairs and empty follicles.
- Loose anagen syndrome is typically seen in young girls with poor adherence of anagen hair to follicle which can be easily pulled out. Trichoscopy shows sparse hair, reduced hair shafts per follicular unit or trichorrhexis nodosa.[32]
- Short Anagen hair syndrome is where the hair does not grow long because of extremely short anagen phase. Trichoscopy shows normal hair density and regrowing hair of different lengths.[33]
- Histopathological examination is rarely needed.
- Hair pull test in late satge shows almost complete telogen hairs as anagen hair are lost. Loose anagen hair syndrome show ruffled hair shaft cuticle due to loss of inner and outer root sheath during extraction.[29]

Tinea Capitis

- It is a fungal infection of the hair and scalp, commonly affecting children caused by dermatophyte genera Trichophyton and Microsporum.[34]
- Clinically, it can be non-inflammatory, inflammatory or favus type. Non-inflammatory tinea capitis may again be of grey patch, black dot, alopecia areata-like, seborrheic dermatitis- like or glabrous types. Inflammatory tinea capitis may be of kerion, abscess, pustular or agminate folliculitis types. Favus is caused by *Trichophyton schoenleinii* and is characterized by scutula with centrally emerging hair follicle.[(35)]
- Morphologically, it can be of 3 types: endothrix where hair shaft is filled with fungal hyphae and arthroconidia with weakening of all hair shaft elements, ectothrix where fungal hyphae and arthroconidia cover outside of hair upto the zone of keratinization and may damage cuticle and favus where there is formation of air spaces within the infected hair shaft.[36]
- Trichoscopic features of tinea capitis include comma hair, cockscrew hair, interrupted/ morse code hair, block hairs, i-hairs, zig zag hairs, black dots and elongated blood vessels. Yellow amorphous areas and wax coloured perifollicular areas are seen in favus.[37]

- Comma hair and cockscrew hair are characteristic and are due to damage of hair shaft or cuticle by fungal elements. Cockscrew hairs have multiple coils and twists in contrast to comma hair.
- Morse code hair show multiple interrupted transverse bands regularly distributed through the hair shaft.
- Zig zag hairs are sharply bent at multiple points and may fracture easily at the site of bending.
- Short hair with distal hyperpigmented zone preceded by thin hypopigmented band are called i-hair.
- Short distal hair with no accentuation are called block hairs.
- Scalp scrapings and hair root examination are used to confirm the diagnosis of tinea capitis. Two types of patterns may be seen on potassium hydroxide examination of these specimens- an endothrix pattern and an ectothrix pattern. Endothrix spores are formed within the hair shaft and cuticle is intact here and this type of sporulation is seen in *Trichophyton violaceum, Trichophyton tonsurans, Trichophyton schoenleinii* and *Trichophyton simii*. Ectothrix spores are seen on the outer surface of shaft and here the cuticle is destroyed and this type of sporulation is commonly seen in *Microsporum canis, Microsporum audouinii, Microsporum ferruginium* and *Trichophyton*

mentagrophytes. Trichophyton rubrum is capable of giving rise to both the sporulations.

- Treatment of tinea capitis is essentially systemic. Griseofulvin given in the dose of 10-20 mg/kg body weight daily for a period of 3 months, terbinafine 5mg/kg daily given for a period of 4 weeks, itraconazole 5mg/kg daily for 6 weeks or ketoconazole 200mg/day for 12 weeks. Pulse therapy may also be given for treatment of tinea capitis. Terbinafine 5mg/kg or itraconazole 5mg/kg daily can be given 7 days. This constitutes a single pulse. A second pulse may be given 2 weeks after the first pulse and a third pulse may be given after 3 weeks of the second pulse. Topical selenium sulphide shampoo, ketoconazole shampoo or zinc pyrithinone containing shampoo may be used to reduce spore load.[(1)]
- Scarring alopecia following inflammatory tinea capitis is common. It is resistant to treatment and usually irreversible.

Trichotillomania

- It is form of traction alopecia resulting from repeated removal of one's own hair.
- Diagnostic criteria according to DSM-IV (Diagnostic and statistical manual of Mental disorders) include: [38]
    - Recurrent pulling of own hair leading to hair loss
    - Increasing sense of tension immediately before pulling out hair or when attempting to resist behavior
    - Pleasure, gratification or relief when pulling out hair
    - Hair pulling cannot be attributed to any other mental disorder
    - Significant impairment in social or occupational functioning
- It commonly affects females between 9 and 13 years of age and commonly affects vertex where hair loss with "tonsure trichotillomania" or "Friar Tuck sign" is seen.[39]
- Trichoscopy shows decreased hair density, hair shafts broken at different lengths, trichoptilosis (hairs with split ends), irregularly coiled hair, upright regrowing hair and black dots. Flame hairs, amorphous hair residues, yellow dots with black peppering, tulip hairs and V-sign may be seen. Rarely, Microexclamation mark hairs are seen.

- Flame hairs are semi-transparent, wavy and cone shaped hair residues resembling flame.[41]
- V-Sign is created when two or more hairs emerging from one follicular unit are pulled simultaneously and break at the same length above the scalp surface.
- Histopathology shows normal density of hair follicles. Distorted hair follicles without inflammation is the diagnostic finding. “Torn away” hair and “hamburger sign” (Vertically oriented split in hair shaft with protinaceous material and erythrocytes) are also specific. Pigment casts and trichomalacia are also seen due to hair follicle trauma.[42]
- The mainstay of treatment for trichotillomania includes behavioural therapy. Parents must be asked to support children during stressful events. Drugs which like tricyclic antidepressants and selective serotonin reuptake inhibitors may be used for treatment.[1]

Traction Alopecia

- It occurs due to unintentional hair traction due to social, cultural or hair styling procedures.
- Clinically, it may be marginal or non-marginal, with initial perifollicular erythema and hair thinning followed, by perifollicular scarring and scarring alopecia.
- Trichoscopy of traction alopecia is similar to trichotillomania but features like white areas lacking hair follicles may be seen in late stage due to scarring.[43]
- Histopathology is similar to trichotillomania but more subtle. Late stage shows scarring of hair follicles known as “follicular casts”. [29]
- Treatment of tractional alopecia is avoidance of traction or tight brading of hair. However, once the alopecia becomes a scarring one, reversal is not possible.[(1)]

CICATRICIAL ALOPECIA

Lichen Planopilaris

- Lichen planopilaris is the most common cause of primary scarring alopecia in adults. It commonly affects vertex but may involve any part of the scalp. Early lesions include violaceous perifollicular erythema and perifollicular keratotic lesions. These ultimately heal with perifollicular atrophic scar which may merge with each other.[44]
- Trichoscopy in active lesion shows perifollicular scaling, tubular scales entangling the hair shaft, hair casts, violaceous areas and elongated linear blood vessels. Inactive lesions show irregular, large white dots, tufted hairs, milky red and white areas.
- The most characteristic feature is perifollicular scaling which migrate for 2-3mm above the hair shaft to form a “collar-like” or “tubular” perifollicular hyperkeratosis.[45] Perifollicular scaling sourrounding a pilosebaceous unit with single hair is seen in patients with lichen planopilaris.
- Some authors have used the term strawberry ice-cream to describe the colour of early cicatricial alopecia of lichen planopilaris.
- Perifollicular blue-gray surrounding empty hair follicles have been seen dark phototypes. This occurs due to supepidermal melanin,

produced in the hair follicles, that entered catagen phase before definite involution, resulting in end stage fibrotic tracts.[45, 46]

- White dots in lichen planopilaris are due to perifollicular fibrosis. They are large and irregular compared to the pin point white dots due to empty follicles and merge to form ivory white or milky red areas.[47]
- Wickham striae may be seen in lichen planopilaris
- Acquired pili torti may be seen at the periphery of the scarred area.
- Histopathology shows focal bandlike infiltrate around hair follicles near the bulge. Vacuolar degeneration of the basal layer of outer root sheath with follicular plugging and wedge shaped hypergranulosis of infundibulum are seen. Interfollicular epidermis is normal. Late stage shows vertically oriented fibrous tracts which have replaced hair follicles.[48]
- Follicular lichen planus can be managed wuth topical therapies such as steroids and cyclosporine. Systemic therapies include the mainstay oral steroids, oral retinoids, chloroquine, thalidomide, griseofulvin and cyclosporine.[1]

Frontal fibrosing alopecia

- Frontal fibrosing alopecia is a primary lymphocytic cicatricial alopecia. It is thought to be a variant of lichen planopilaris. It is commonly seen in post-menopausal females.[49]
- It is characterized by symmetric hair line recession in frontotemporal or frontoparietal region with loss of eyebrows in most patients. Body hair loss is seen in some patients affecting axilla, pubis or limbs. [50]
- Trichoscopic findings include lack of follicular openings over an ivory-coloured homogenous background. Mild perifollicular erythema & scaling are seen. Perifollicular brown or brown-violet area is seen in dark skinned individuals.[51]
- Eyebrows show multiple regularly distributed red dots or gray dots. [(52)]
- Histopathology shows features similar to lichen planopilaris with perifollicular lymphocytic infiltrate near the bulge and hydropic degeneration of the basal cell. Hair follicles are replaced by fibrous tissue with diffuse scarring in the later stage.[29]
- Frontal fibrosing alopecia can be managed with topical minoxidil, topical or intralesional steroids, systemic retinoids, hydroxychloroquine, griseofulvin and finasteride.[1]

Discoid lupus erythematosus

- Discoid lupus erythematosus is a primary lymphocytic cicatricial alopecia which commonly occurs in females between 20 to 40 years of age. The early lesion is a erythematous discoid alopecia patch with follicular plugging and adherent scaling. The lesions may be hypopigmented or hyperpigmented. Late lesions are atrophic white plaques without follicular ostia.[53]
- Trichoscopic examination of early lesions show thick arborizing vessels, large yellow dots, fine interfollicular scaling, scattered brown discolouration, red dots or blue gray dots. Prefibrotic lesions show spider vessels in yellow dots. Late lesions show white areas with loss of follicular orfices & arborizing vessels.[45,52]
- Large yellow dots are due to follicular plugging whereas dark brown “dirty”pigmentation is due to pigment incontinence. Red dots correspond to widened infundibula with dense perivascular lymphocytic infiltrate, dilated vessels with extravasated erythrocytes.
- Histopathology shows hyperkeratosis, follicular plugging, atrophic epidermis, basal cell degeneration, patchy infiltrate around hair follicle bulge, civatte bodies and pigment incontinence around the hair follicle. Interfollicular epidermis may also be involved. In the burnt out stage, diffuse fibrosis with loss of hair follicles is seen.[54]

- Topical steroids and intralesional steroids are the mainstay of treatment for limited discoid lupus erythematosus.
- Other topical treatments include topical tacrolimus (0.1%), tazarotene (0.05%) and topical imiquimod (5%). Intralesional IFN-α has been used for treating discoid lupus erythematosus.
- In cases of rapid progression or extensive involvement, systemic therapy must be given. This includes antimalarials like chloroquine and hydroxychloroquine, oral steroids, isotretinoin, dapsone, clofazimine, methotrexate, azathioprine, mychophenolate mofetil, systemic IFN-α and monoclonal anti-CD4 antibodies. [1]
- Discoid lupus erythematosus may progress to systemic lupus erythematosus. This is seen especially in generalized variety of discoid lupus erythematosus.

Folliculitis decalvans

- Folliculitis decalvans is a neutophilic primary cicatricial alopecia which primarily involves the vertex and the occipital scalp.[55] The characteristic feature is presence of multiple hairs emerging from a single dilated hair follicle opening due to clustering of adjacent follicular openings as a result of fibrosis and retention of telogen hairs within the involved follicles.[56]
- It is characterized by recurrent follicular pustules with erythema, dark yellow-grey scales, haemorrhagic crusts, erosions around hair follicles in the active phase which lead to irregularly shaped patches of scarring alopecia [57]
- Tufted folliculitis is thought to be a variant of folliculitis decalvans but with smaller areas being affected, minimal hair loss and better prognosis.[44]
- In the active disease trichoscopic findings include tufts of 5 or more hairs in one follicular unit, yellow follicular pustules, yellowish tubular scaling with collar formation, yellowish discharge, starburst sign, folds of epidermal hyperplasia and elongated, coiled or lace like vessels. Inactive longstanding disease shows folds of epidermal hyperplasia, milky red areas and white areas lacking follicular opening. [45, 52]

- The follicular pustules develop in the following stages:
    - Stage 1:Yellowish perifollicular discolouration with slight bulge
    - Stage 2: Yellowish perifollicular discolouration with visible bulge and mild blood extravasation
    - Stage 3:Dark red perifollicular discoloration
- The characterisitic feature occurring in 60% of cases of folliculitis decalvans is tubular scaling which is also seen in lichen planopilaris but the ones in folliculitis decalvans are yellow and frequently roll away from the hair shaft at the distal end to form collar like structures. [52] These may detach from the scalp surface and move outwards with the growing hair.
- Starburst sign is seen in 66% of patients where fold of epidermal hyperplasia with hyperkeratosis surrounds follicular units containing hair tufts.
- Histopathological examination reveals abscess at level of follicular infundibulum which may be dialated. Later lesions show predominant lymphocytic infiltrate with few plasma cells, eosinophils, neutrophils and giant cells. Hyperkeratosis, follicular plugging and tufting may be present. Later stage shows diffuse dermal fibrosis. [29]

- The treatment in folliculitis decalvans is based on pus culture and sensitivity. Cotrimoxazole, ciprofloxacin, clindamycin or clarithromycin in combination with high dose rifampicin for 10 weeks. Topical antibiotics like 1% mupirocin, 1% fusidic acid or 2% erythromycin may be prescribed. Recurrences are extremely common. Oral steroids and oral retinoids are other medications which have been tried in this condition. [1]
- The treatment of this condition is seldom satisfactory with frequent recurrences. The end result of is the destruction of the stem cells in the bulge region with irreversible scarring alopecia.
- The tufted folliculitis variant of folliculitis decalvans is more localized and milder in its course with less frequent recurrences and less scarring.

Dissecting cellulitis

- Dissecting folliculitis is a chronic, progressive, inflammatory disease that occurs commonly in young adults especially men.[44]
- It starts as follicular occlusion on the vertex or occiput, followed by perifollicular pustules, nodules, abscesses with interconnecting sinuses.[58]

- It may be associated with acne congloblata, hidradenitis suppurativa and pilonidal sinuses, together called follicular occlusion tetrad.
- Trichoscopic findings of active dissecting cellulitis include yellow structureless areas, yellow soap bubble/3D dots with or without hair shafts, black dots, pinpoint-like vessels with whitish halo, cutaneous clefts with emerging hairs and hair tufts.[45,52]
- Earliest finding trichoscopically is non-specific large perifollicular pustules.
- 3D soap bubble dots are yellow structureless areas with or without hair follicles. They are seen in 100% of patients.
- Irregular violaceous areas corresponding to areas of pigment incontinence may also be seen.
- Cutaneous clefts with small hair tufts are also specific for dissecting cellulitis.
- Late stage shows irregular areas of white fibrosis.
- Histopathology shows a perifollicular heavy infiltrate with lymphocytes, histiocytes and polymorphonuclear cells. Pilosebaceous abscess formation occurs followed by hair follicle destruction and finally fibrosis. Sometimes a granulomatous response may be seen.[59]

- Treatment of this condition includes exclusion of the other associated follicular occlusion dependent conditions like acne congloblata, hidadrenitis suppurativa, pilonidal sinus and pyoderma gangrenosum.
- The mainstay among drugs for treatment includes oral isotretinoin therapy. It is started at a dose of 1 mg/kg body weight daily for minimum of four months and then tapered to 0.75mg/kg continued for 6 months. The other modalities of treatment include tetracycline, steroids, dapsone, colchicine, minocycline, cyproterone acetate. Topical isotretinoin, topical antibiotics and intralesional steroids may be used for treatment. Laser epilation using Q-switched ruby laser or diode laser may be done to curb inflammatory process.[1]

Pseudopelade of Brocq

- It is a primary scarring lymphocytic alopecia which commonly affects middle aged females.[60]
- It is characterized clinically by asymptomatic, non-inflamed porcelain white, small irregular patches located in central scalp area imitating "footprints in the snow" appearance. The course is slowly progressive.[45,52]
- The trichoscopic findings are non-specific including absent hair follicles over smooth white areas with dystrophic hairs present at the

periphery of the lesion. No features suggestive of other cicatricial alopecia are seen.

- Histopathology shows coloumns of fibrosis which replace hair follicle associated with sebaceous gland atrophy. Epidermis and sweat glands are normal with absence of marked inflammation in the dermis. Initially, mild perivascular or perifollicular (around infundibulum or mid-follicle) lymphocytic infiltrate is seen which is replaced by patchy infiltrate later on. The infiltrate disappears when the stage of diffuse fibrosis sets in.[29]

Aims And Objectives

AIMS & OBJECTIVES

1. To study the dermoscopic findings in non-cicatricial & cicatricial alopecias.
2. To study the correlation between dermoscopic findings & histopathological examination, in cases of alopecia with doubtful diagnosis.
3. To study the correlation between dermoscopic findings & scraping with KOH examination in suspected cases of tinea capitis.

Materials And Methods

MATERIALS AND METHODS

STUDY CENTER : Department of Dermatology,
Madras Medical College & Rajiv Gandhi
Government General Hospital, Chennai-03

STUDY DESIGN : Prospective, observational study

DURATION OF STUDY: MAY, 2013- APRIL, 2014

INCLUSION CRITERIA: Patients above the age of 2 months with alopecia who are ready to give consent (parental consent in case of minors) attending Department of Dermatology, RGGGH were included in the study.

EXCLUSION CRITERIA: Children below the age of 2 months, patients not willing to give consent, patients on treatment for more than 3 months were excluded from the study.

METHODOLOGY: Detailed and informed consent was obtained from the patients enrolled in the study. Detailed clinical history, in terms of, onset, duration & progression of alopecia, associated skin lesions, drug intake, other medical illnesses, stress factors was obtained from the patients. Clinical examination to assess type of alopecia, hair pull test and coudability sign were performed in all cases. Dermoscopic examination was done to assess

density of hair over different regions of the scalp, hair shaft diameter variations, hair shaft defects, erythema & scaling, atrophy, loss of follicular orifices, presence of yellow dots, black dots, white dots, capillary loop changes & telengectasia, presence of vellus hair & depigmented hair, and also to assess presence of other specific findings for various alopecias.

Scraping with KOH examination was done in suspected cases of tinea capitis & scalp biopsy with histopathological examination using Haematoxylin & Eosin was done in cases with doubtful diagnosis.

Observation And Results

OBSERVATIONS & RESULTS

82 patients with alopecia attended the Dermatology OPD of Madras Medical College between 1st May, 2013 and 30 April, 2014. Non-cicatricial alopecia accounted for 59 cases and cicatricial alopecia was found in 19 patients. 4 patients had congenital alopecia.

The main non-scarring alopecia was alopecia areata (24.39%), followed by tinea capitis (17.07%), androgenetic alopecia(12.20%), telogen effluvium (8.54%), trichotillomania (7.32%) and anagen effluvium (2.44%).

The main scarring alopecia were discoid lupus erythematosus and lichen planopilaris which accounted for 7 cases each (8.54%). They were followed by folliculitis decalvans (2.44%), pseudopelade of Brocq (2.44%) and frontal fibrosing alopecia (1.22%).

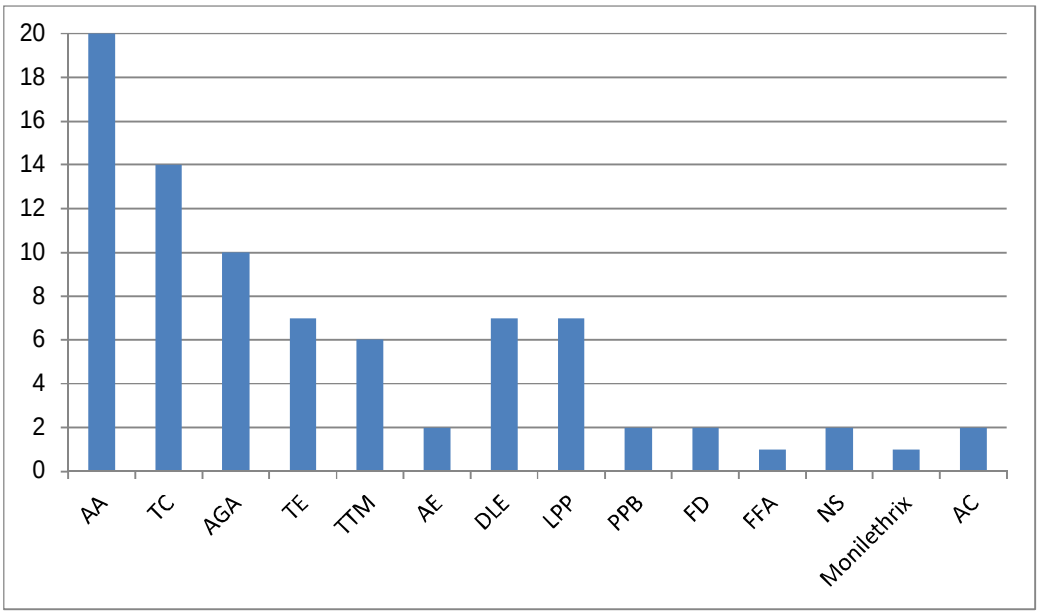
There were 2 cases of nevus sebaceous (2.44%) and one each of aplasia cutis and monilethrix(1.22%).

The following table depicts the incidence of various alopecia during the study.

TABLE 1: DISTRIBUTION OF PATIENTS ON BASIS OF ETIOLOGICAL DIAGNOSIS

DIAGNOSIS	NUMBER OF PATIENTS (n=82)	PERCENTAGE (%)
Alopecia areata (AA)	20	24.39
Tinea capitis (TC)	14	17.07
Androgenetic alopecia (AGA)	10	12.20
Telogen effluvium (TE)	7	8.54
Trichotillomania (TTM)	6	7.32
Anagen effluvium (AE)	2	2.44
Discoid lupus erythematosus (DLE)	7	8.54
Lichen planopilaris (LPP)	7	8.54
Pseudopelade of Brocq (PPB)	2	2.44
Folliculitis decalvans (FD)	2	2.44
Frontal fibrosing alopecia (FFA)	1	1.22
Nevus sebaceous (NS)	2	2.44
Aplasia cutis (AC)	1	1.22
Hair shaft disorder (monilethrix)	1	1.22

FIGURE 1: DISTRIBUTION OF PATIENTS ON BASIS OF ETIOLOGICAL DIAGNOSIS



Out of the 82 patients, 44 (53.66%) were male and 38 (46.34%) were female. The number of paediatric patients (<13 years) was 26(31.71%).

ALOPECIA AREATA

Alopecia areata accounted for 20 patients. This included 11 male and 9 female patients. 7 patients were in the paediatric age group (<13 years). All the patients had non-scarring alopecia with 13 showing positive hair pull test.

TABLE 2: DERMOSCOPIC FINDINGS IN ALOPECIA AREATA

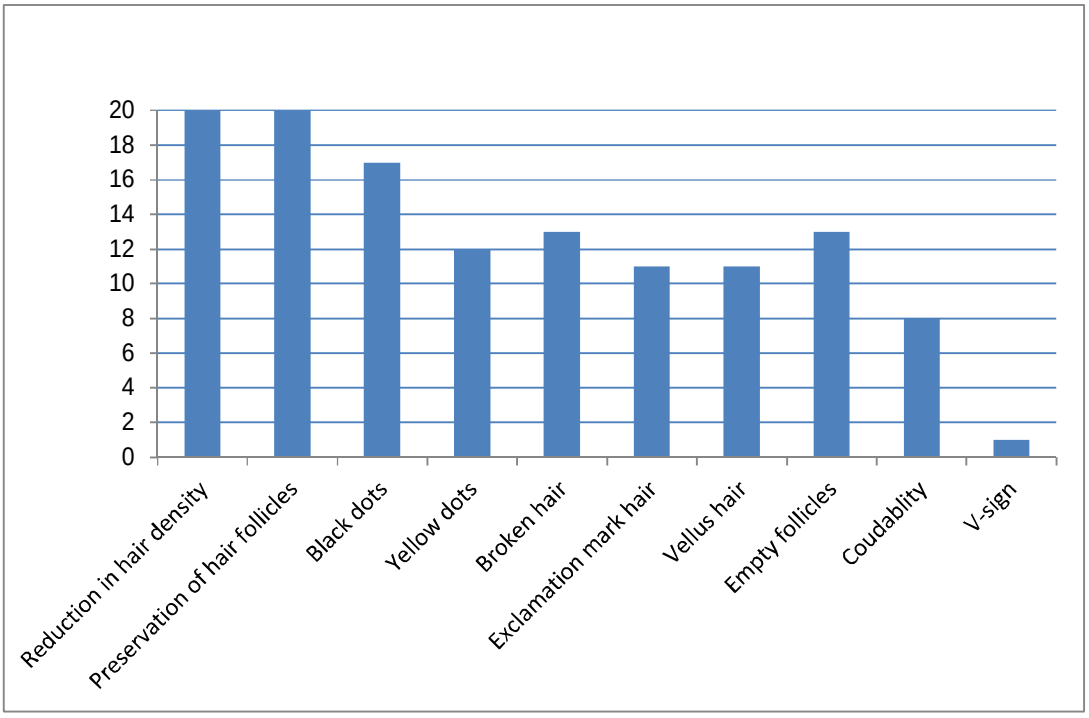
DERMOSCOPIC FINDING	NUMBER OF PATIENTS WITH FINDING (n=20)	PERCENTAGE (%)
Reduction in hair density	20	100
Preservation of hair follicles	20	100
Black dots	17	85
Yellow dots	12	60
Broken hair	13	65
Exclamation mark hair	11	55
Vellus hair	11	55
Empty follicles	13	65
Coudablity	8	40
V-sign	1	5

Dermoscopically, all patients had preserved hair follicles. Black dots (85%) and broken hairs (65%) were the commonest dermoscopic finding followed by empty follicles (65%), yellow dots (60%), exclamation mark hair

(55%), vellus hair (55%) and coudability sign (40%). V-Sign was seen in only one patient.

Clinically, exclamation mark hair and coudability sign were seen in in 3(15%) and 6 (30%) patients respectively whereas dermoscopically, exclamation mark hair and coudability sign were seen in 11(55%) and 8(40%) patients respectively.

FIGURE 2: DERMOSCOPIC FINDINGS IN ALOPECIA AREATA



Out of the 20 patients with alopecia areata, 16(80%) were of patchy type, 3(15%) were extensive type (1 alopecia totalis, 1 alopecia universalis, 1 diffuse alopecia areata) and 1 (5%) ophiasis type.

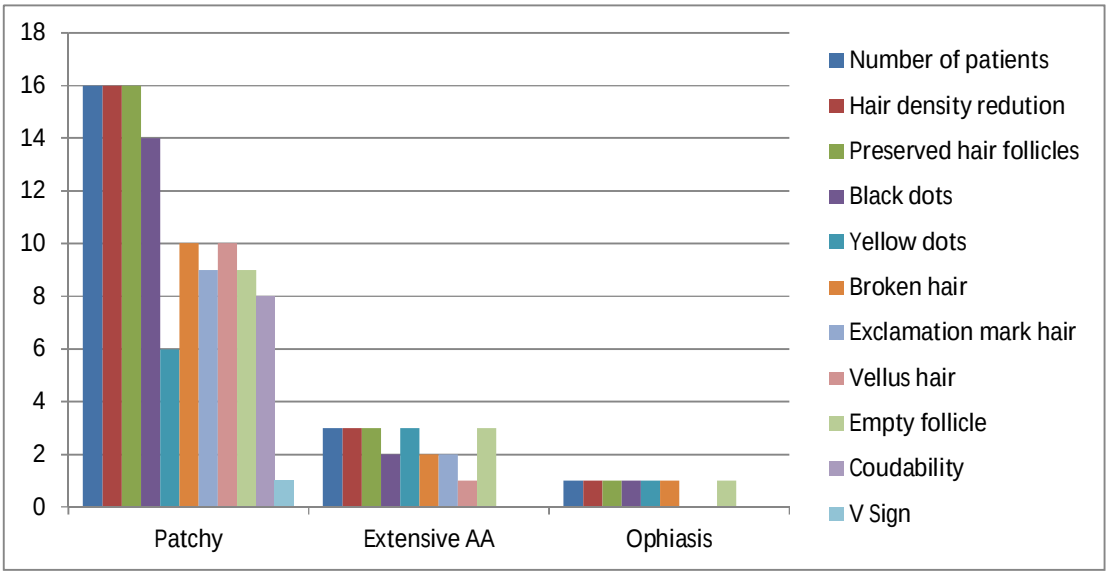
TABLE 3: DERMOSCOPIC FINDINGS IN DIFFERENT TYPES OF ALOPECIA AREATA

DERMOSCOPIC FINDING	Patchy AA	Extensive AA	Ophiasis
Number of patients	16	3	1
Reduction in hair density	16	3	1
Preservation of hair follicles	16	3	1
Black dots	14	2	1
Yellow dots	6	3	1
Broken hair	10	2	1
Exclamation mark hair	9	2	-
Vellus hair	10	1	-
Empty follicles	9	3	1
Coudablity	8	-	-
V-sign	1	-	-

Dermoscopic findings in all types of alopecia areata are similar with black dots, broken hair and yellow dots being the commonest findings in all the three subtypes. Yellow dots and empty follicles were seen in all cases of extensive alopecia areata. Dermoscopic coudablity and V-sign were seen in

patchy alopecia areata. Dermoscopic exclamation mark hair and vellus hair were not seen in the single case of ophiasis alopecia areata.

FIGURE 3: DERMOSCOPIC FINDINGS IN DIFFERENT TYPES OF ALOPECIA AREATA



Scalp scraping and hair root examination for fungal elements were negative in all cases.

TINEA CAPITIS

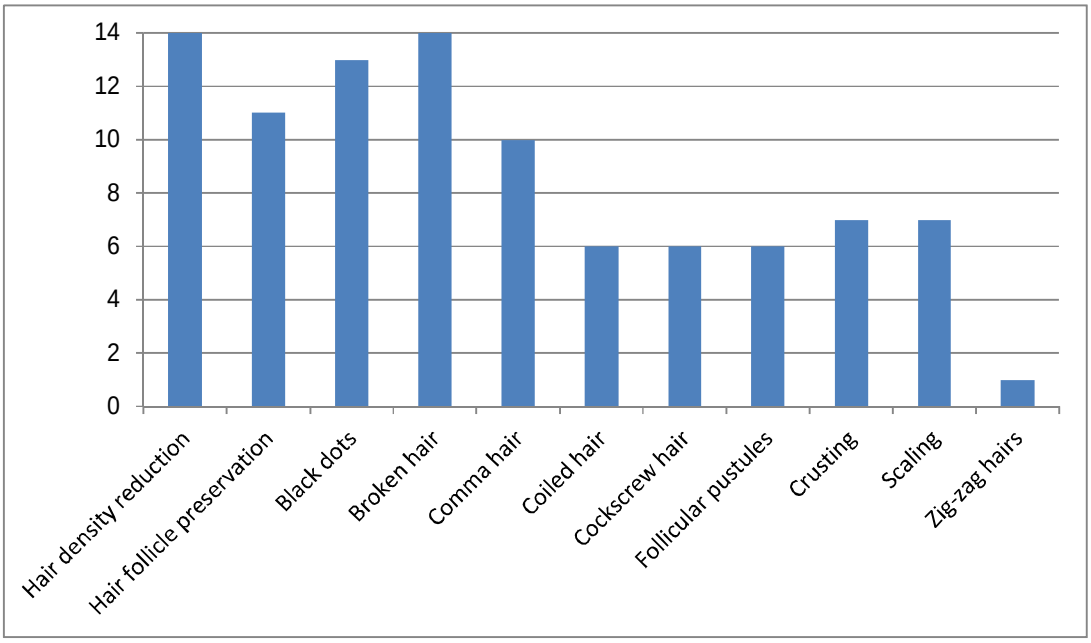
14 patients presented with Tinea capitis during the study period. This included 9 male and 5 female patients. 11 patients were in the paediatric age group (<13 years). None of the patients showed evidence of scarring and 11 of them had a positive hair pull test.

TABLE 4: DERMOSCOPIC FINDINGS IN TINEA CAPITIS

DERMOSCOPIC FINDINGS	NUMBER OF PATIENTS (n=14)	PERCENTAGE (%)
Hair density reduction	14	100
Hair follicle preservation	11 (3 patients- Hair follicles obscured)	78.57
Black dots	13	92.86
Broken hair	14	100
Comma hair	10	71.43
Coiled hair	6	42.86
Cockscrew hair	6	42.86
Follicular pustules	6	42.86
Crusting	7	50.00
Scaling	7	50.00
Zig-zag hairs	1	7.14

Dermoscopically, the commonest finding in cases of tinea capitis was broken hairs (100%) and black dots (92.86%). This was followed by comma hair (71.43%), crusting(50%), scaling (50%), coiled hair (42.86%), cockscrew hair (42.86%) and follicular pustules (42.86%). Zig-zag hair were seen in 1 patient.

FIGURE 4: DERMOSCOPIC FINDINGS IN TINEA CAPITIS



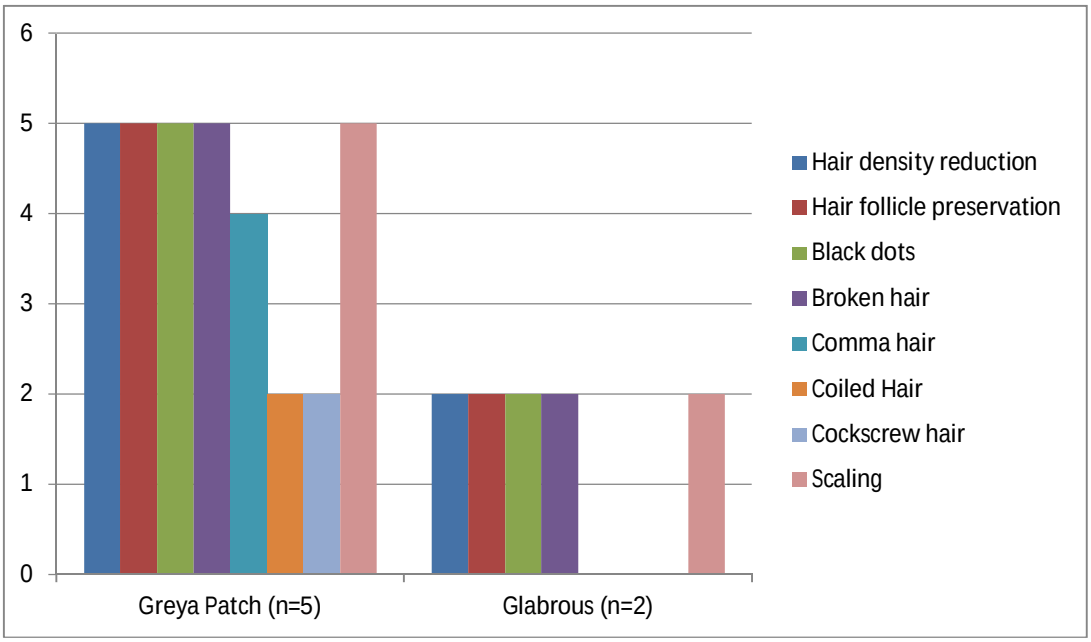
Out of the 14 patients, 7(50%) had non-inflammatory and 7(50%) had inflammatory type of tinea capitis. This included 5 patients with grey patch, 2 with glabrous type, 3 patients each of kerion and pustular types and 1 with incognito type.

TABLE 5: DERMOSCOPIC FINDINGS IN NON-INLAMMATORY TYPES OF TINEA CAPITIS

DERMOSCOPIC FINDINGS	Grey patch (n=5)	Glabrous (n=2)	Non-inflammatory (n=7)
Hair density reduction	5	2	7
Hair follicle preservation	5	2	7
Black dots	5	2	7
Broken hair	5	2	7
Comma hair	4	-	4
Coiled hair	2	-	2
Cockscrew hair	2	-	2
Scaling	5	2	7

The dermoscopic findings in non-inflammatory type consisted mainly of black dots, broken hair and scaling. Comma hair, coiled hair and cockscrew hairs were not seen in the glabrous type.

FIGURE 6: DERMOSCOPIC FINDINGS IN NON-INFLAMMATORY TINEA CAPITIS



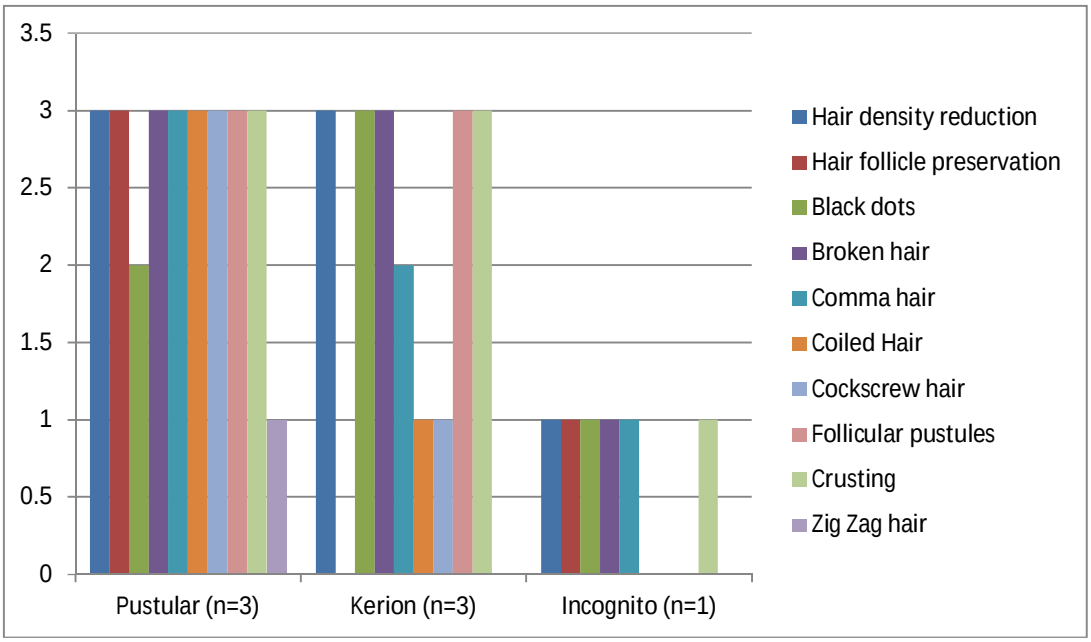
Dermoscopic findings in case of inflammatory tinea capitis found black dots, broken hair, comma hair, follicular pustules and crusting to be present in most cases. Cockscrew hair and coiled hair were commonly seen in Pustular type of tinea capitis. Zig-zag hairs were seen in a single case of pustular tinea capitis.

TABLE 7: DERMOSCOPIC FINDINGS IN INFLAMMATORY TYPE OF TINEA CAPITIS

DERMOSCOPIC FINDINGS	Pustular (n=3)	Kerion (n=3)	Incognito (n=1)	Inflammatory (n=7)
Hair density reduction	3	3	1	7
Hair follicle preservation	3	-	1	4
Black dots	2	3	1	6
Broken hair	3	3	1	7
Comma hair	3	2	1	6
Coiled hair	3	1	-	4
Cockscrew hair	3	1	-	4
Follicular pustules	3	3	-	6
Crusting	3	3	1	7
Zig-zag hairs	1	-	-	1

Comma hair & cockscrew hair, the specific findings were found in most but not all cases. Black dots, broken hair, follicular pustules, crusting and scaling were non-specific but common findings.

FIGURE 7: DERMOSCOPIC FINDINGS IN INFLAMMATORY TINEA CAPITIS



Scalp scarping and hair root examination in all cases showed fungal elements in form of refractile arthrospores spores in and around the hair follicle. Thus, the dermoscopic diagnosis of tinea capitis was consistent with mycological examination in all cases (100%).

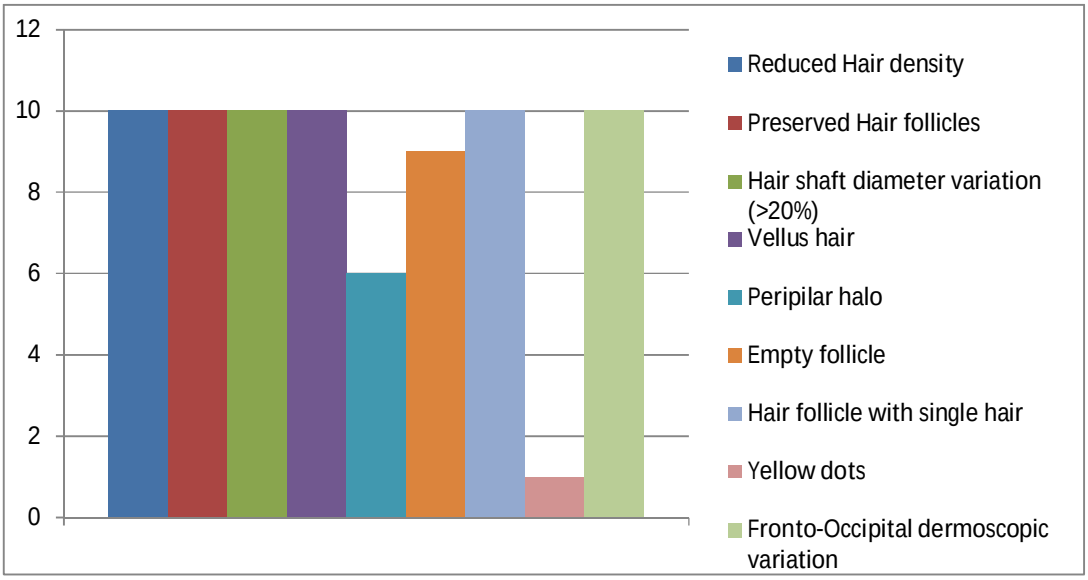
ANDROGENETIC ALOPECIA

10 patients with androgenetic alopecia were seen during study period which included 6 males and 4 females. All patients were between 20 years and 40 years of age. Clinically, all patients had non-scarring alopecia.

TABLE 8: DERMOSCOPIC FINDINGS IN ANDROGENETIC ALOPECIA

DERMOSCOPIC FINDINGS	NUMBER OF PATIENTS (n=10)	PERCENTAGE (%)
Reduced hair density	10	100
Preserved hair follicles	10	100
Hair shaft diameter variations (>20%)	10	100
Vellus hair	10	100
Peripliar halo sign	6	60
Empty follicles	9	90
Hair follicles with single hair (>35%)	10	100
Yellow dots	1	10
Fronto-occiital dermoscopic variation	10	100

FIGURE 8: DERMOSCOPIC FINDINGS IN ANDROGENETIC ALOPECIA



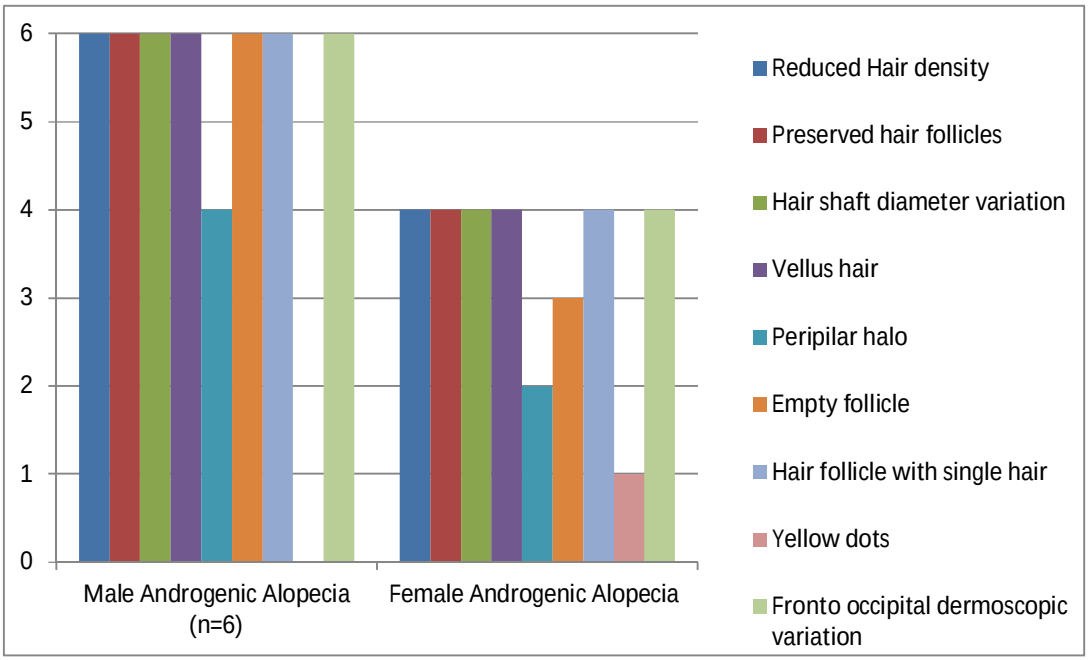
Dermoscopic findings in androgenetic alopecia included hair shaft diameter variation (>20%), vellus hair and hair follicles with single hair follicle (>35%) in all cases. Empty follicles were seen in most patients (90%) with peripilar halo being the other finding (60%). Yellow dots were seen only in one patient (10%). The findings were noted in the frontal region and absent in the occipital region.

TABLE 9: DERMOSCOPIC FINDINGS IN MALE AND FEMALE ANDROGENETIC ALOPECIA

DERMOSCOPIC FINDINGS	MALE (n=6)	FEMALE (n=4)
Reduced hair density	6	4
Preserved hair follicles	6	4
Hair shaft diameter variations (>20%)	6	4
Vellus hair	6	4
Peripliar halo sign	4	2
Empty follicles	6	3
Hair follicles with single hair (>35%)	6	4
Yellow dots	-	1
Fronto-occiital dermoscopic variation	6	4

Dermoscopic findings in both male and female patients are similar with hair shaft diameter variation (>20%), vellus hairs, hair follicles with single hair (>35%) & empty follicles being the commonest findings.

FIGURE 9: DERMOSCOPIC FINDINGS IN MALE AND FEMALE ANDROGENETIC ALOPECIA



Both male and female patients showed majority of dermoscopic findings in the frontal region with absence of findings in the occipital region.

TELOGEN EFFLUVIUM

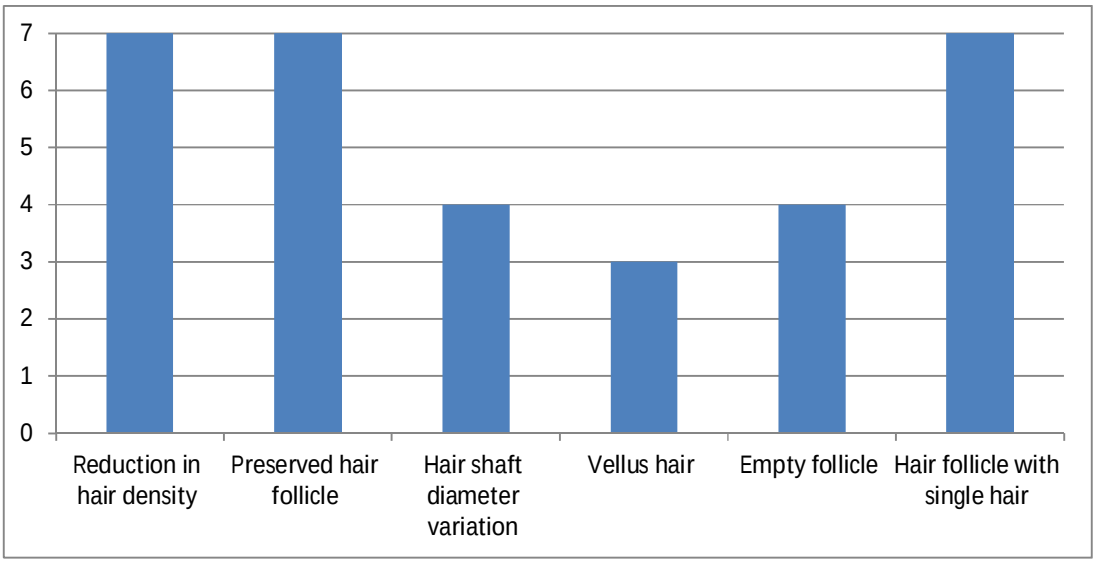
7 patients of telogen effluvium included 6 females and 1 male. All the patients were between 20 years and 34 years of age.

TABLE 10: DERMOSCOPIC FINDINGS IN TELOGEN EFFLUVIUM

DERMOSCOPIC FINDINGS	NUMBER OF PATIENTS (n=7)	PERCENTAGE (%)
Reduction in hair density	7	100
Preservation of hair follicles	7	100
Hair shaft diameter variation (<20%)	4	57.14
Vellus hair	3	42.86
Peripilar halo sign	-	-
Empty hair follicles	4	57.14
Hair follicle with single hairs (>35%)	7	100
Yellow dots	-	-
Fronto-occipital dermoscopic variation	-	-

Dermoscopy in telogen effluvium showed preservation of hair follicles in all cases. Hair follicles with single hair (>35%) were seen in all cases. Hair shaft diameter variation (<20%) (57%) and empty follicles (57%) were the other common findings. No case with peripliar halo or yellow dots was seen. Patient showed no fronto-occipital variation in dermoscopy findings.

FIGURE 10: DERMOSCOPIC FINDINGS IN TELLOGEN EFFLUVIUM



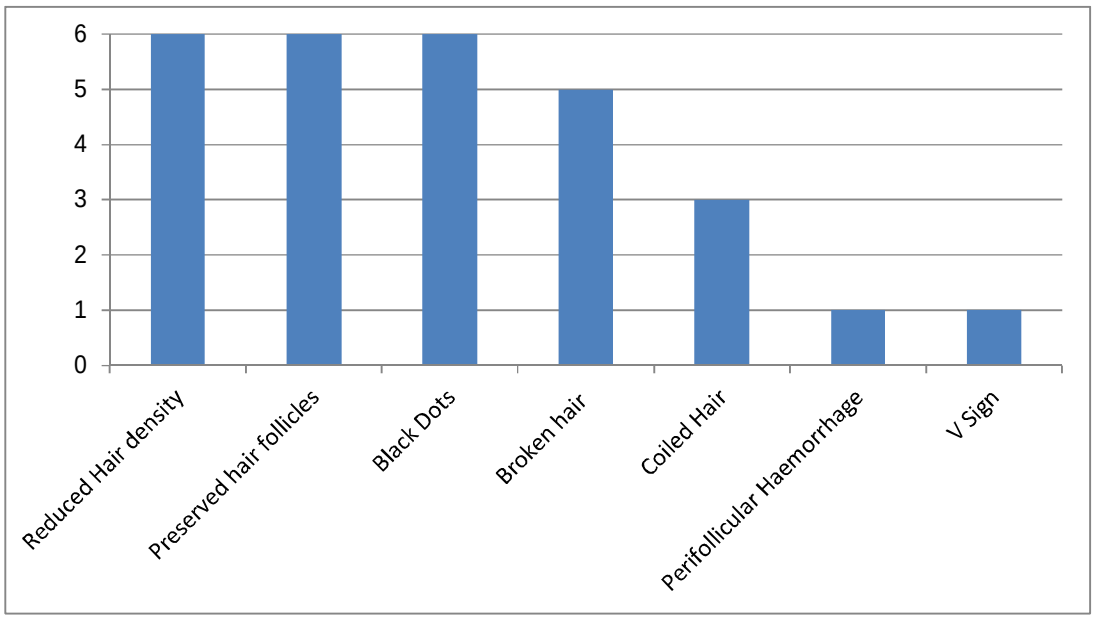
TRICHOTILOMANIA

6 cases of trichotillomania attended the Dermatology OPD during the study period. This included 4 females and 2 males. 4 out of the 6 patients were of paediatric age group (<13 years). Clinically, all the cases were of non-scarring type with 5 showing a positive hair pull test.

TABLE 11 : DERMOSCOPIC FINDINGS IN TRICHOTILLOMANIA

DERMOSCOPIC FINDING	NUMBER OF PATIENTS (n=6)	PERCENTAGE (%)
Reduced hair density	6	100
Preserved hair follicles	6	100
Black dots	6	100
Broken hair (Varying length)	5	83.33
Coiled hair	3	50.00
Perifollicular haemorrhage	1	16.67
V- Sign	1	16.67

FIGURE 11: DERMOSCOPIC FINDINGS IN TRICHOTILLOMANIA



Dermoscopically, all cases showed preserved hair follicles with black dots (100%) and broken hair at irregular lengths being the commonest finding (83.33%). Coiled hair (50%), perifollicular haemorrhage (16.67%) and V-sign (16.67%) were the other findings.

Scalp Scraping & hair root examination for fungal elements in all patients was negative.

ANAGEN EFFLUVIUM

During the study period, 2 patients with anagen effluvium both on chemotherapy for 2-4 weeks presented to our department. Both had clinically non-scarring hair loss which was diffuse in nature. Both the patients showed a positive hair pull test.

TABLE 12: DERMOSCOPIC FINDINGS IN ANAGEN EFFLUVIUM

DERMOSCOPIC FINDING	NUMBER OF PATIENTS (n=2)	PERCENTAGE (%)
Reduced hair density	2	100
Preserved hair follicles	2	100
Short terminal hair	2	100
Abruptly constricted hair	2	100
Perifollicular brown pigmentation	1	50
Empty follicle	2	100
Hair shaft breakage	2	100

Dermoscopic examination showed short terminal hair, abruptly constricted hair, numerous empty follicles and hair shaft breakage in both cases (100%). Perifollicular brown pigmentation was seen in 1 patient (50%).

LICHEN PLANOPILARIS

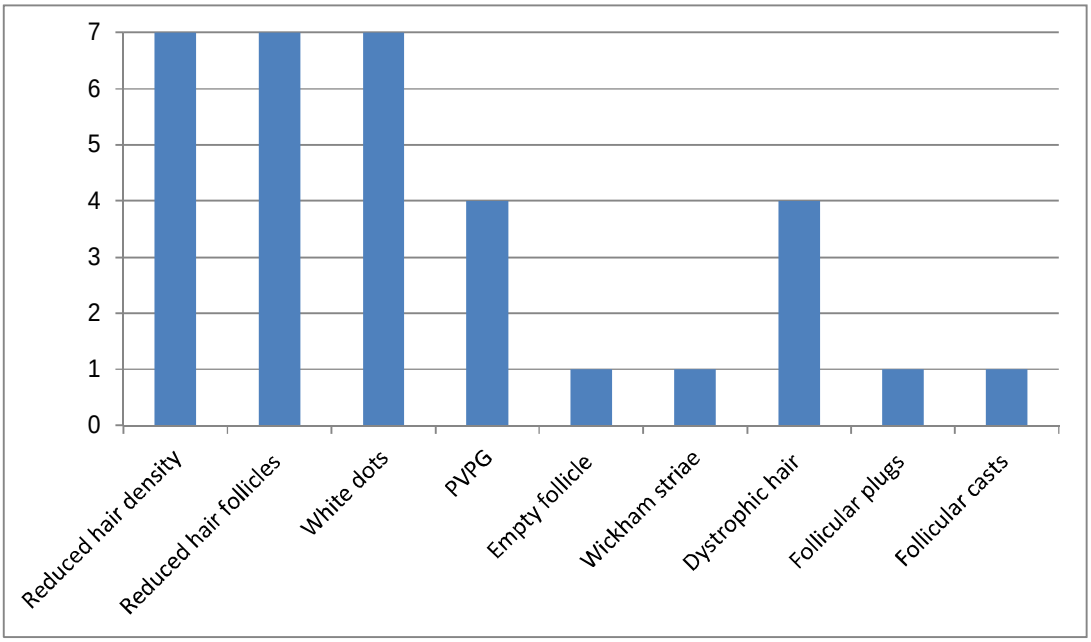
7 patients with lichen palnopilaris were seen during the study period. This included 3 males and 4 females. The patients were between the age group of 20 to 50 years. Clinically, all patients showed scarring alopecia. Two patients showed associated violaceous buccal mucosal pigmentation and 1 patient showed typical plaques of lichen planus over the body.

TABLE 13: DERMOSCOPIC FINDINGS IN LICHENPLANOPILARIS

DERMOSCOPIC FINDINGS	NUMBER OF PATIENTS	PERCENTAGE(%)
Reduced hair density	7	100
Reduced hair follicles	7	100
White dots	7	100
Perifollicular violaceous pigmentation	4	57.14
Empty follicles	1	14.29
Wickham striae	1	14.29
Dystrophic hair	4	57.14
Follicular plugs	1	14.29
Follicular casts	1	14.29

All the patients showed dermoscopic reduction in hair follicles with white dots being the commones dermoscopic finding seen in all patients (100%). Perifollicular violaceous pigmentation and dystrophic hair were the other common finding (57.14%). Wickham's striae, empty follicles, follicular plugs and follicular casts were seen in 1 patient each (14.29%).

FIGURE 12: DERMOSCOPIC FINDINGS IN LICHEN PLANOPILARIS



Biopsy and histopathologic examination were done in 6 out 7 patients. They were consistent with lichen planopilaris and included follicular plugging, hypergranulosis of infundibulum, basal cell degeneration of outer root sheet, pigment incontinence and perifollicular mononuclear infiltrate.

DISCOID LUPUS ERYTHEMATOSUS

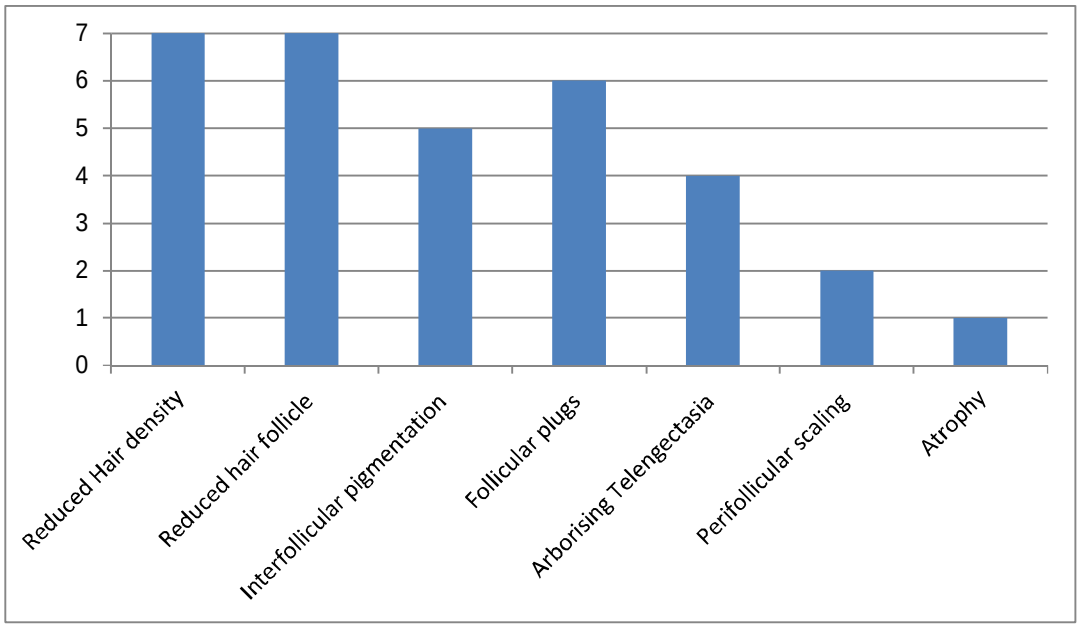
7 patients with discoid lupus erythematosus included 6 female patients and 1 male patient. All patients were between 20 and 40 years of age and showed scarring alopecia clinically. 4 patients had associated photosensitivity and 3 patients had discoid lesions over the body.

TABLE 14: DERMOSCOPIC FINDINGS IN DISCOID LUPUS ERYTHEMATOSUS

DERMOSCOPIC FINDINGS	NUMBER OF PATIENTS (n=7)	PERCENTAGE (%)
Reduced hair density	7	100
Reduced hair follicles	7	100
Interfollicular pigmentation	5	71.43
Follicular plugs	6	85.71
Telengectasia	4	57.14
Perifollicular scaling	2	28.5
Atrophy	1	14.29

Dermoscopy showed reduced number of hair follicles in all 7 patients (100%). Follicular plugs (85.71%) was the next common finding followed by interfollicular pigmentation (71.43%). Telengectasia (57.14%), perifollicular scaling (28.5%) and atrophy (14.29%) were the other findings seen on dermoscopic examination.

FIGURE 13: DERMOSCOPIC FINDINGS IN DISCOID LUPUS ERYTHEMATOSUS



Hisopathological examination was done in 6 out of 7 patients. Biopsy findings included follicular plugging, basal cell degeneration of outer root sheet, pigment incontinence, civatte bodies, perifollicular mononuclear inflammatory infiltrate and interfollicular epidermal atrophy consistent with discoid lupus erythematosus.

PSEUDOPELADE OF BROCQ

2 female patients in the age group between 35 and 40 years presented with Pseudopelade of Brocq. Both the patients had scarring alopecia clinically.

TABLE 15: DERMOSCOPIC FINDINGS IN PSEUDOPELADE OF BROCQ

DERMOSCOPIC FINDING	NUMBER OF PATIENTS (n=2)	PERCENTAGE (%)
Reduction in hair density	2	100
Reduced hair follicles	2	100
Perifollicular brown pigmentation	1	50
Ivory white areas	1	50
Dystrophic hair	1	50
Empty follicles	1	50

Dermoscopic examination showed reduction in hair follicles (100%) with perifollicular brown pigmentation, ivory white areas, dystrophic hair and empty follicles as the other findings.

Histopathological examination in both patients showed reduction in hair follicles with perifollicular inflammatory infiltrate.

FOLLICULITIS DECALVANS

The study period had 2 patients with folliculitis decalvans presenting in Dermatology OPD. One of the patients was a 23 year female and the other patient was a 34 year male. Both showed scarring alopecia clinically.

TABLE 16: DERMOSCOPIC FINDINGS IN FOLLICUITIS DECALVANS

DERMOSCOPIC FINDING	NUMBER OF PATIENTS (n=2)	PERCENTAGE (%)
Reduced hair density	2	100
Reduced hair follicles	2	100
Follicular pustules (Different stages of development)	2	100
Perifollicular crusting	2	100

Dermoscopy showed follicular pustules in various stages of development- yellow raised lesions, yellow lesions with extravasated blood, haemorrhagic pustules and crusts. Sourrounding areas with prior pustles showed reduced hair follicles.

Histopathologic examination showed non-specific findings in both cases.

FRONTAL FIBROSING ALOPECIA

A single patient with frontal fibrosing alopecia was seen during study period. The patient was a 26 year old female with clinically scarring alopecia involving the frontal area and side burns.

Dermoscopic examination showed reduced hair follicles over the lesion. Cream coloured background with multiple hair follicles with single hair was noted over the lesional area. A single hair with follicular cast was noted over the lesional area.

Biopsy from the area and histopathologic examination revealed paucity of hair follicle with mononuclear infiltrate around hair follicles and arrector pilorus muscle. Fibroblasts with fibrous tissue were also noted in the section.

NEVUS SEBACEOUS

2 male patients presented with with progressively verrucous lesion over scalp with absence of hair since birth. Clinical diagnosis in both cases was nevus sebaceous.

Dermoscopic examination showed absence of hair follicles over the plaque with widespread well demarcated lipid globules in the lesion.

Histopathologic examination showed presence of multiple sebaceous glands in the dermis.

APLASIA CUTIS

A one year male patient with absence of hair over a patch over the scalp since birth with clinically scarring alopecia attended the Dermatology OPD during the study period. The clinical diagnosis was made as aplasia cutis.

Dermoscopy showed absence of hair over the patch with multiple stellate scars over the patch.

Histopathologic examination revealed absence of hair follicles and other appendages in the section with flattening of epidermis with irregularly organised connective tissue in the dermis.

MONILETHRIX

A 2 month old baby with absence of hair over scalp and eyebrows since birth with associated thumb defect in right upper limb.

X-Ray of the limb showed ill-developed 1st metacarpal bone with hypoplastic phalanges of the thumb.

Echocardiogaphy revealed ostium secundum type of atrial septal defect.

Dermoscopic examination of scalp and eyebrows showed multiple short hair with nodal dialation and intermodal constrictions. Hear shaft breakage was noted at the internodal regions. Multiple empty follicles were noted.

Thus, a diagnosis of monilethrix with Holt- Oram syndrome was made.

FIGURE14: CLINICAL TYPES OF ALOPECIA AREATA

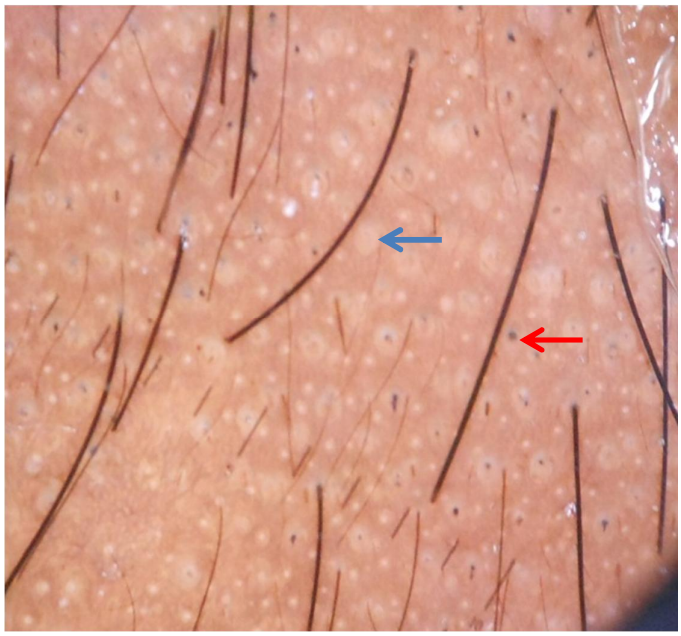
A- PATCHY TYPE

B- ALOPECIA SUBTOTALIS

FIGURE 14: CLINICAL TYPES OF ALOPECIA AREATA

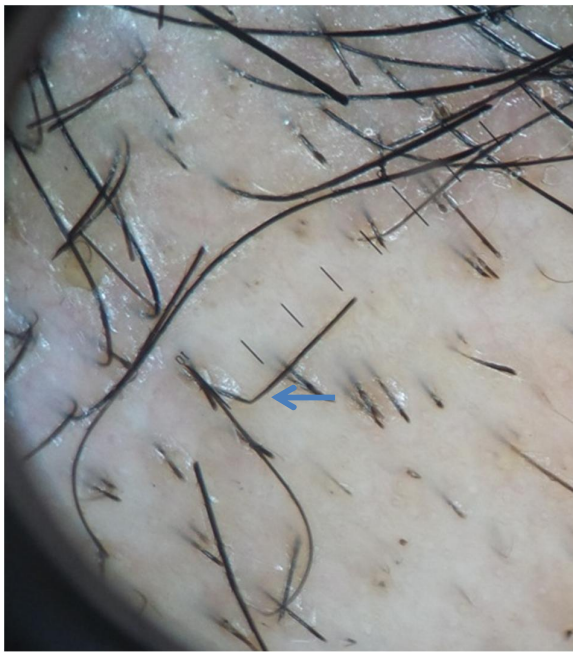
C-OPHIASIS TYPE

FIGURE 15: DERMOSCOPIC FINDINGS IN ALOPECIA AREATA

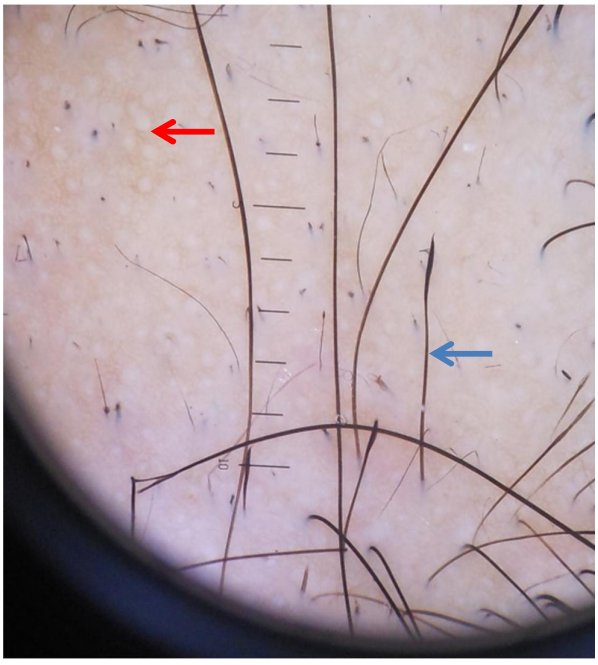


A- Black dots (red arrow) & yellow dots (blue arrow)

FIGURE 15: DERMOSCOPIC FINDINGS IN ALOPECIA AREATA

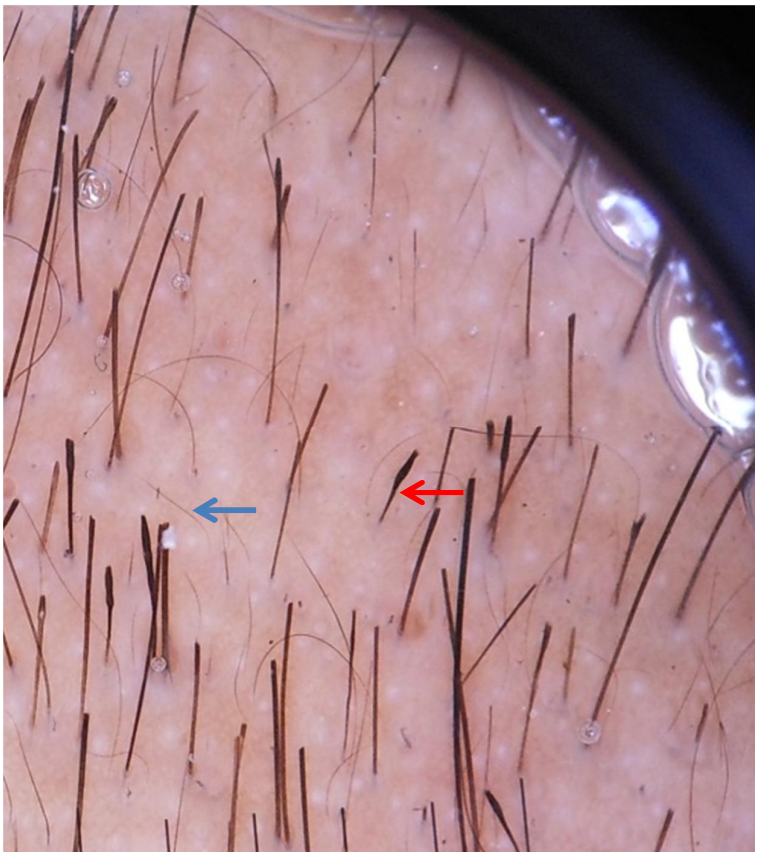


B- Dermoscopic coudability (blue arrow)



C- Exclamation mark hairs (blue arrow), yellow dots (red arrow) & black dots

FIGURE 15: DERMOSCOPIC FINDINGS IN ALOPECIA AREATA



D- Exclamation mark hair (red arrow) & vellus hair (blue arrow)

FIGURE 16: CLINICAL TYPES OF TINEA CAPITIS

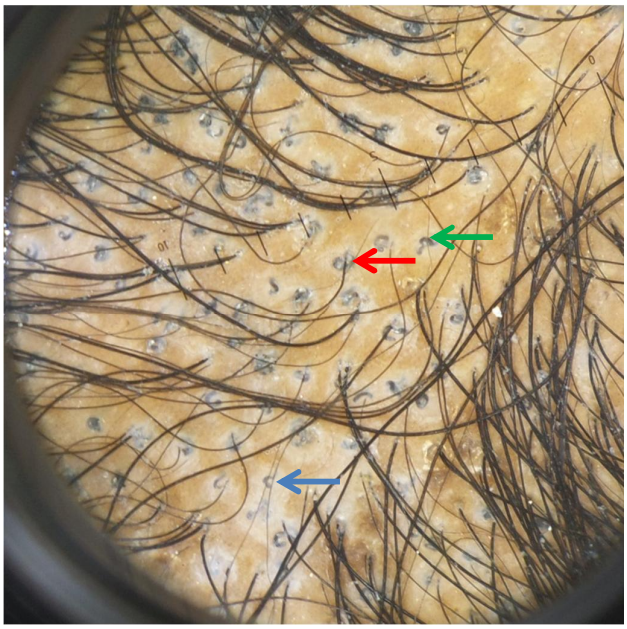
A- Grey patch

B- Pustular

FIGURE 16: CLINICAL TYPES OF TINEA CAPITIS

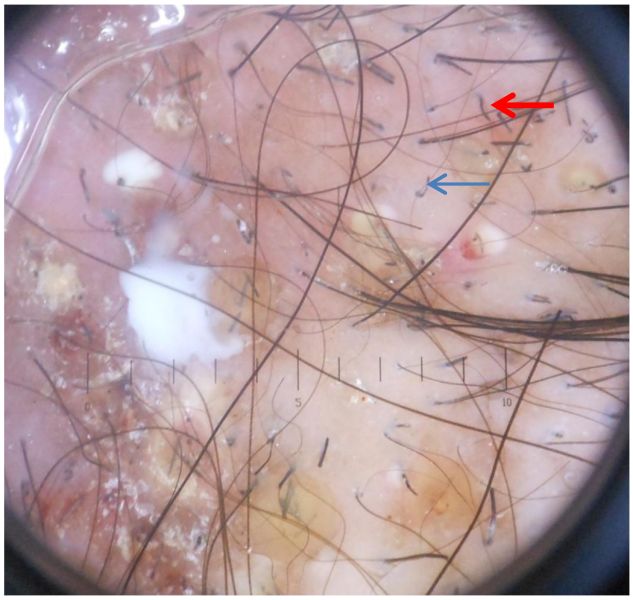
C- Kerion

FIGURE 17: DERMOSCOPIC FINDINGS IN TINEA CAPITIS



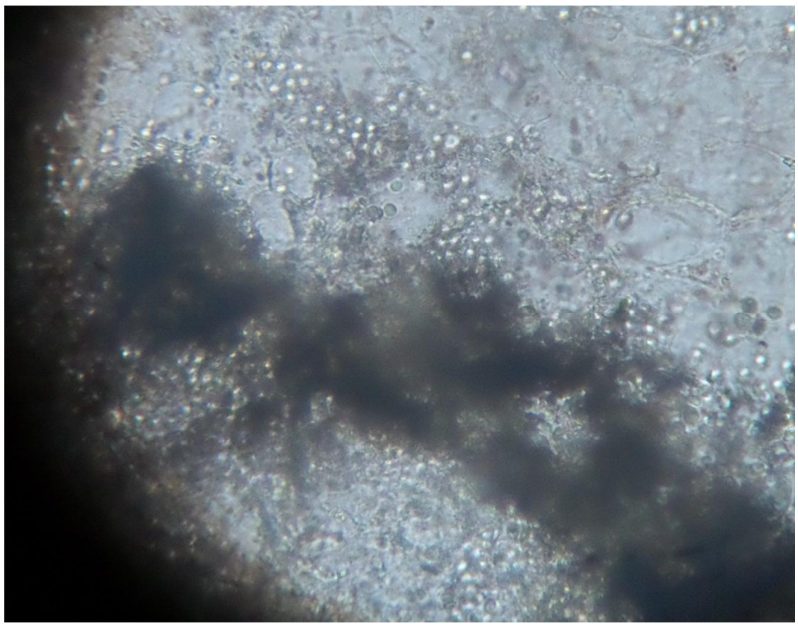
A- Non inflammatory tinea capitis- scaling, comma hair(blue arrow), coiled hair(green arrow) and cockscrew hair(red arrow)

FIGURE 17: DERMOSCOPIC FINDINGS IN TINEA CAPITIS



B- Inflammatory tinea capitis showing follicular pustules, crusting, black dots, broken hair, comma hair(blue arrow) and coiled hair(red arrow)

FIGURE 18: HIGH POWER VIEW OF SCALP SCRAPINGS IN 10% KOH SOLUTION



Multiple refractile arthrospores surrounding hair

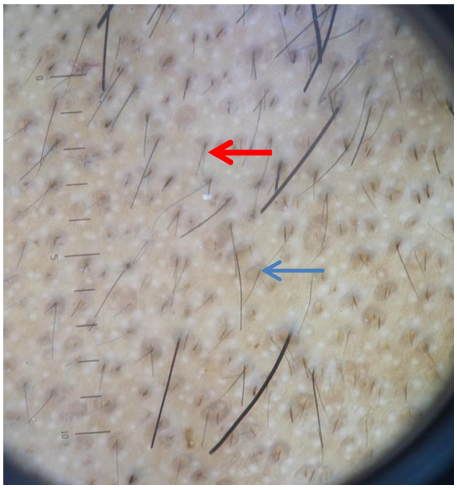
FIGURE 19: CLINICAL PHOTOGRAPHS- ANDROGENETIC ALOPECIA

A- Male Androgenetic Alopecia

B- Female Androgenetic Alopecia

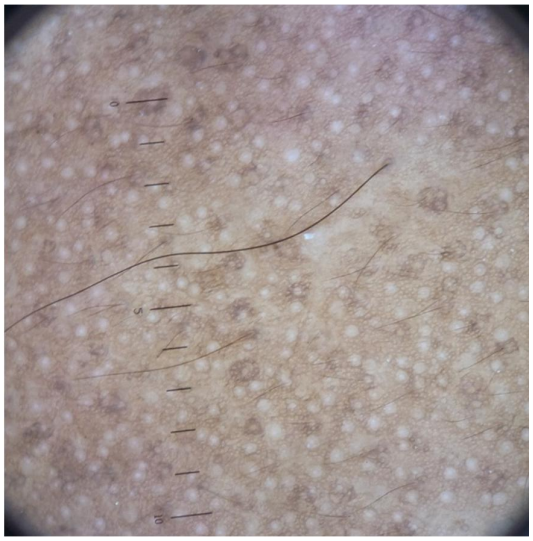
FIGURE 20: DERMOSCOPIC FINDINGS IN ANDROGENETIC ALOPECIA

A- Hair shaft diameter variation (>20%), Hair follicles with single hair (>35%) (blue arrow)



B- Peripilar halo(blue arrow), hair shaft diameter variation (>20%), vellus hair (red arrow)

FIGURE 20: DERMOSCOPIC FINDINGS IN ANDROGENETIC ALOPECIA



C- Vellus hair and empty follicles

FIGURE 21: CLINICAL PHOTOGRAPH- TELOGEN EFFLUVIUM

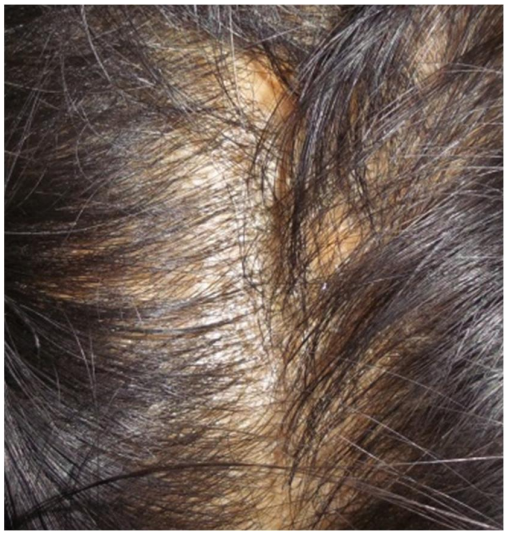
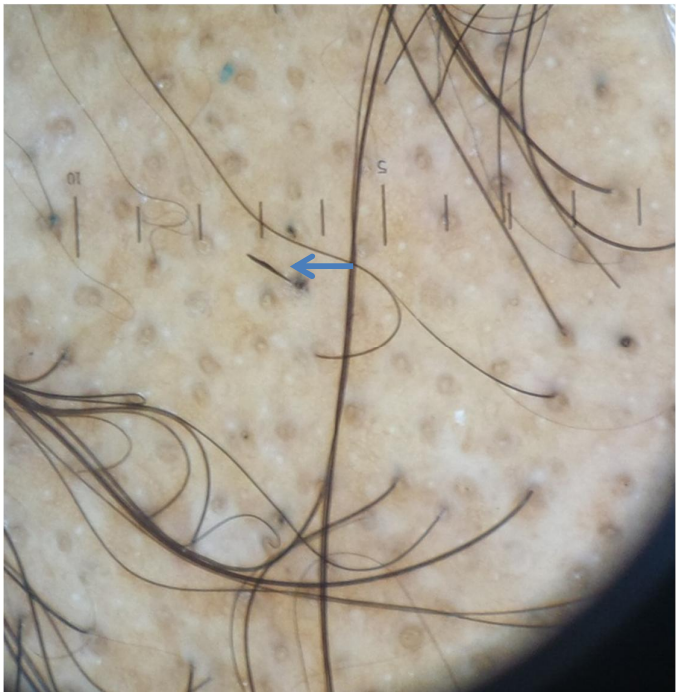


FIGURE 22: DERMOSCOPIC FINDINGS IN TELOGEN EFFLUVIUM

Minimum Hair shaft diameter variation with numerous hair follicles with single hair(>35%)

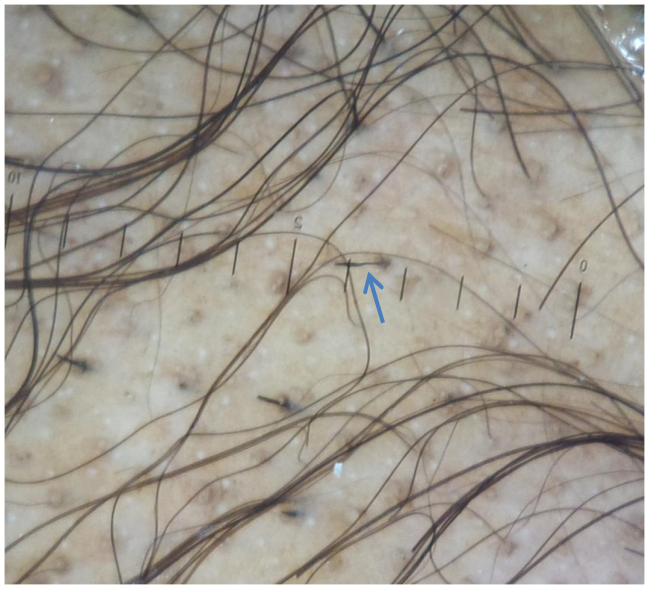
FIGURE 23: CLINICAL PHOTOGRAPH- ANAGEN EFFLUVIUM

FIGURE 24: DERMOSCOPIC FINDINGS: ANAGEN EFFLUVIUM



A- Empty follicles, abruptly constricted terminal hair(blue arrow) and black dots

FIGURE 24: DERMOSCOPIC FINDINGS: ANAGEN EFFLUVIUM



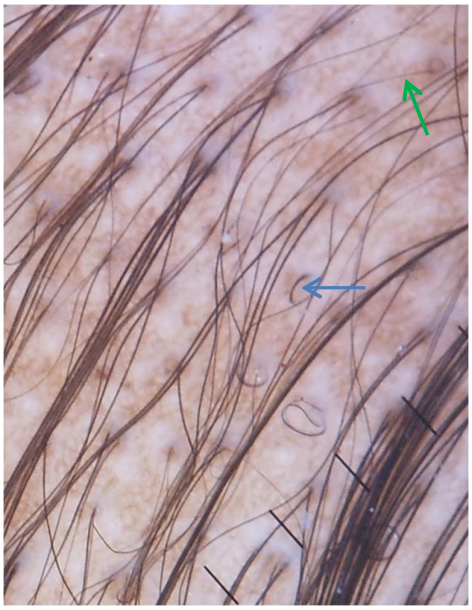
B- Empty follicles, abruptly tapering hair(blue arrow) and black dots

FIGURE 25: CLINICAL PHOTOGRAPH- TRICHOTILOMANIA

FIGURE 26: DERMOSCOPIC FINDINGS IN TRICHOTILOMANIA

A- Black dots, broken hair at irregular lengths, V-sign(green arrow), perifollicular pigmentation(blue arrow)

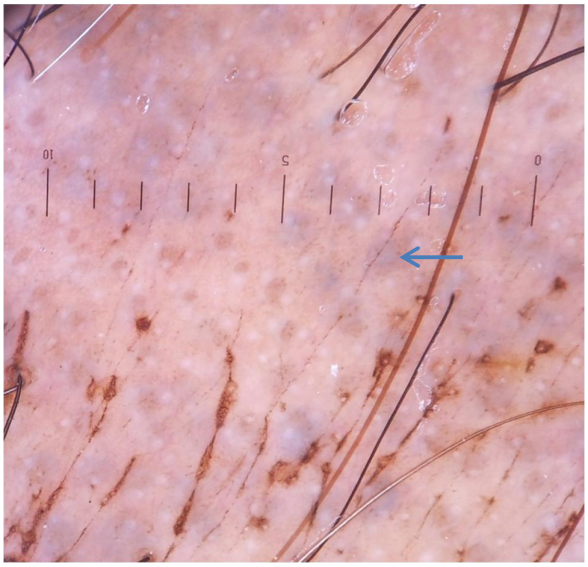
FIGURE 26: DERMOSCOPIC FINDINGS IN TRICHOTILOMANIA



B- Coiled hair (blue arrow) & perifollicular haemorrhage (green arrow)

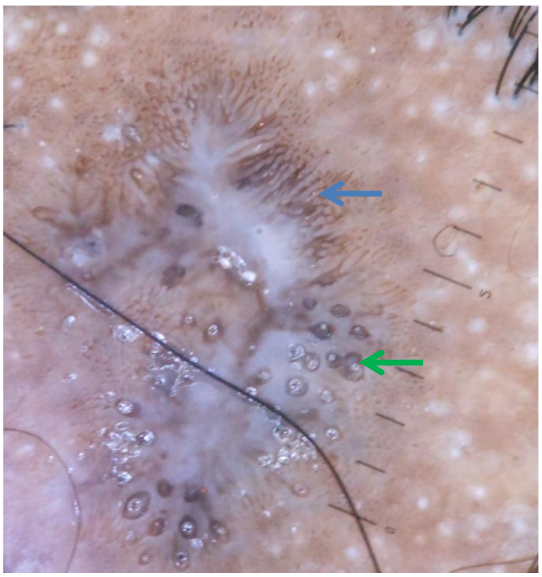
FIGURE 27: CLINICAL PHOTOGRAPH- LICHEN PLANOPILARIS

FIGURE 28: DERMOSCOPIC FINDINGS IN LICHEN PLANOPILARIS



A- Perifollicular violaceous pigmentation(blue arrow)

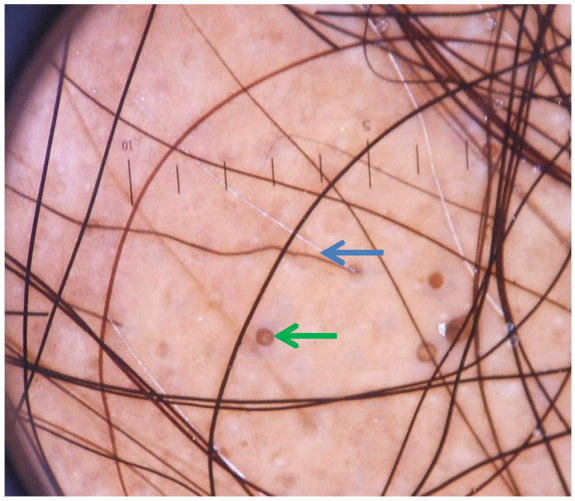
FIGURE 29: DERMOSCOPIC FINDINGS IN LICHEN PLANOPILARIS



B- Wickham striae(blue arrow) and pigment globules(green arrow)

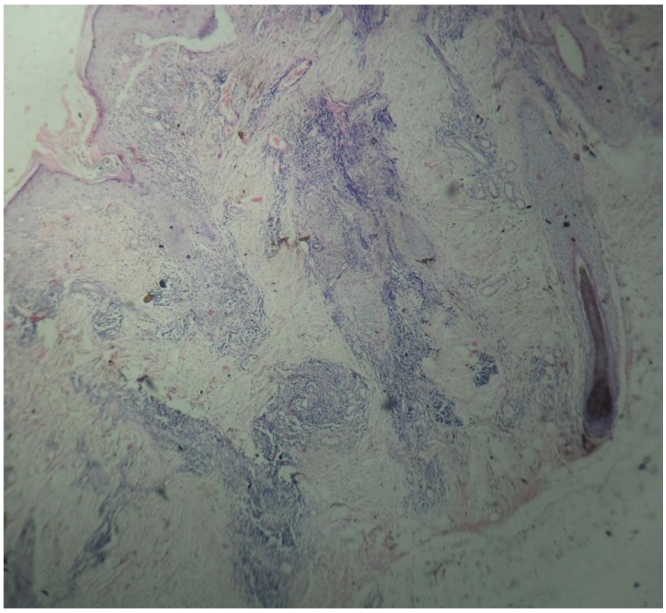
C- Follicular casts

FIGURE 29: DERMOSCOPIC FINDINGS IN LICHEN PLANOPILARIS

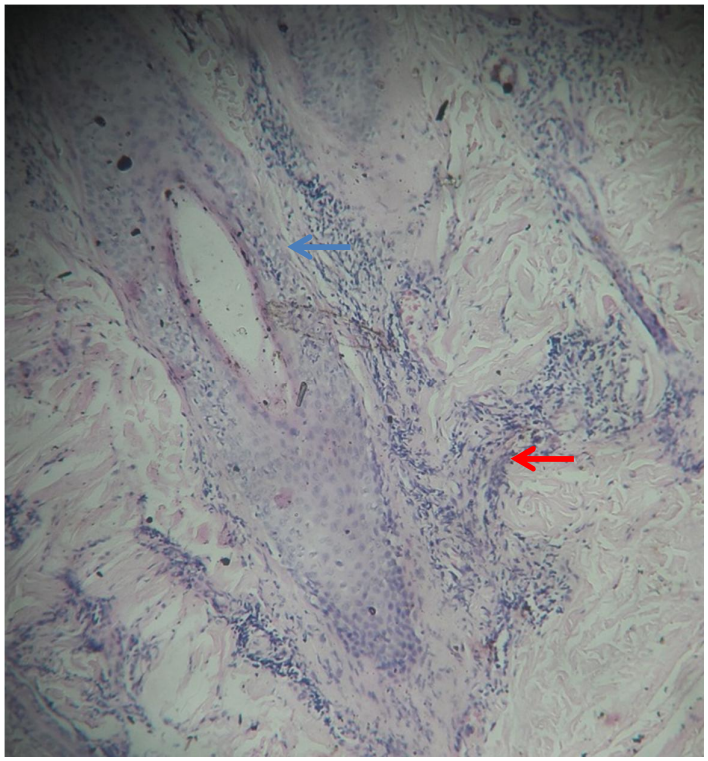


D- Dystrophic hair(blue arrow) and follicular plugs(green arrow)

FIGURE 30: HISTOPATHOLOGY IN LICHEN PLANOPILARIS



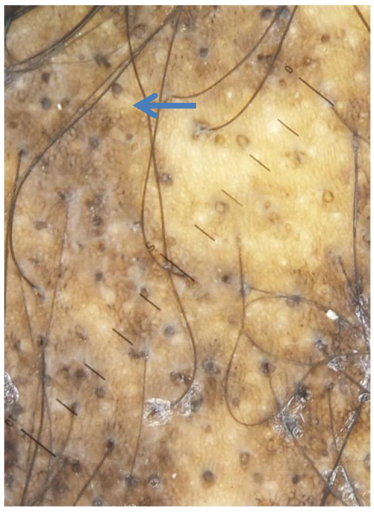
A- **Low power view**- Multiple hair follicles surrounded by perifollicular infiltrate



B- **High power view**- Basal cell degeneration of outer root sheath(blue arrow) with perifollicular monuclear inflammatory infiltrate (red arrow)

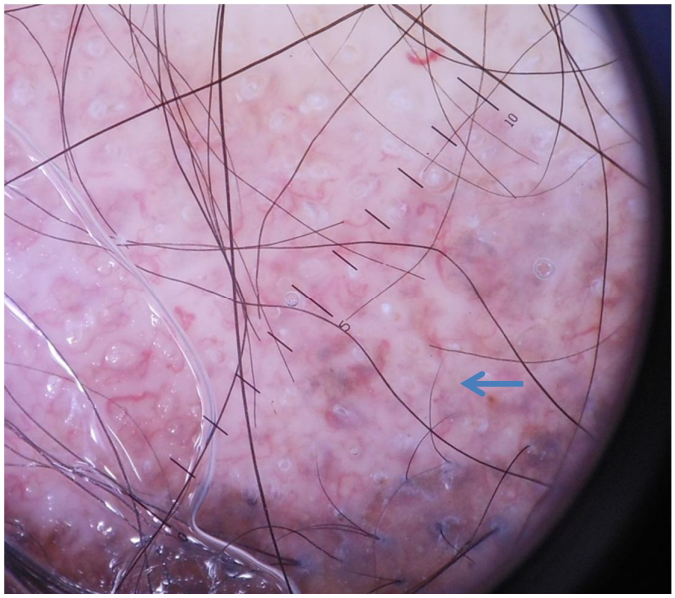
FIGURE 31: CLINICAL PHOTOGRAPH- DISCOID LUPUS ERYTHEMATOSUS

FIGURE 32: DERMOSCOPIC FINDINGS IN DISCOID LUPUS ERYTHEMATOSUS

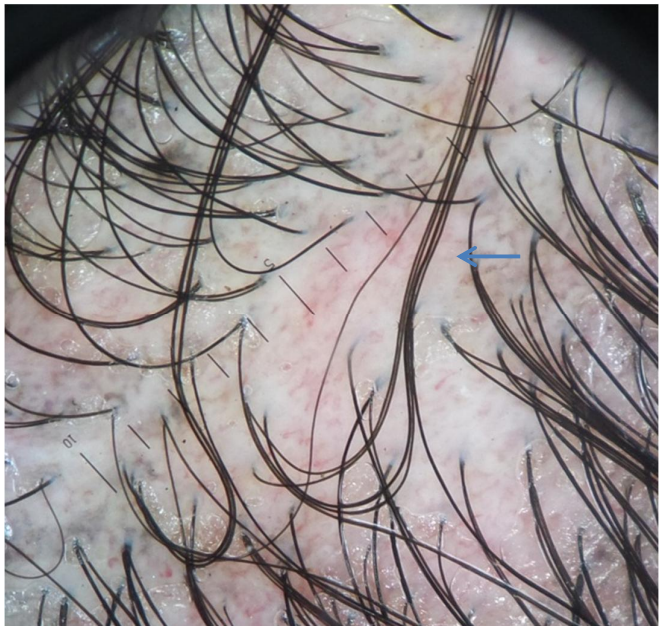


A- Follicular plugs(blue arrow) and interfollicular brown pigmentation

FIGURE 32: DERMOSCOPIC FINDINGS IN DISCOID LUPUS ERYTHEMATOSUS

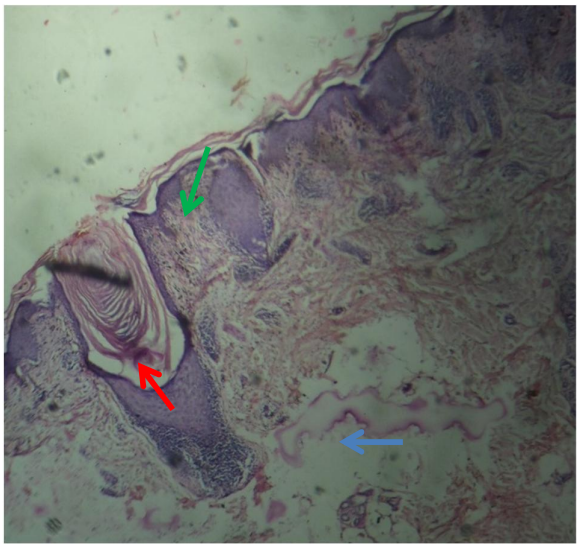


B- Telengectasia(blue arrow) and interfollicular pigmentation

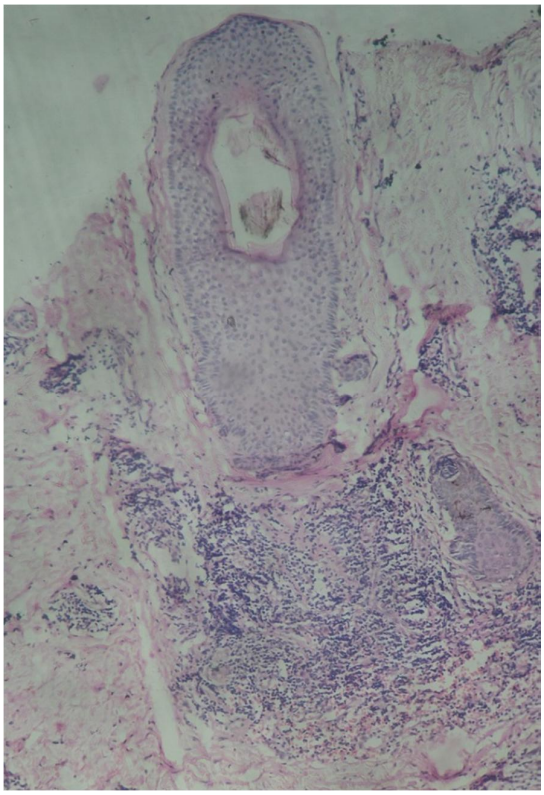


C- Telengectasia(blue arrow) and interfollicular pigmentation

FIGURE 33: HISTOPATHOLOGY IN DISCOID LUPUS ERYTHEMATOSUS



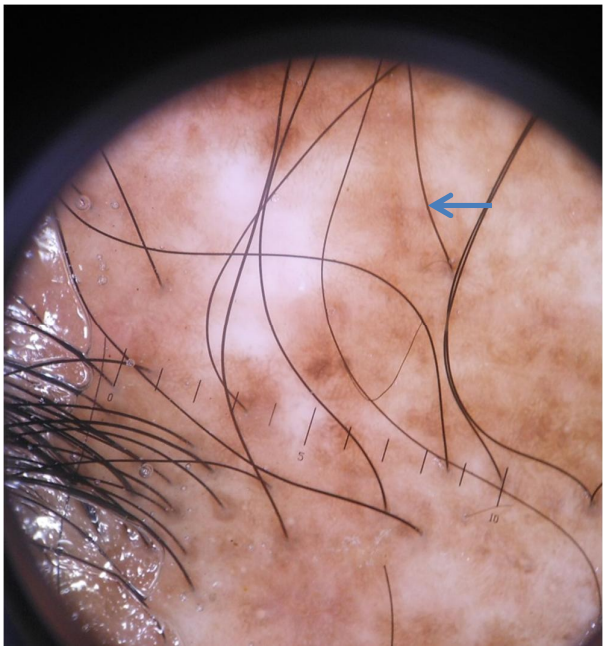
A- **Low power view**- Follicular plugging(green arrow), interfollicular epidermal atrophy(red arrow) and perifollicular inflammatory infiltrate(blue arrow)



B- **High power view**- Patchy perifollicular mononuclear inflammatory infiltrate around hair follicle

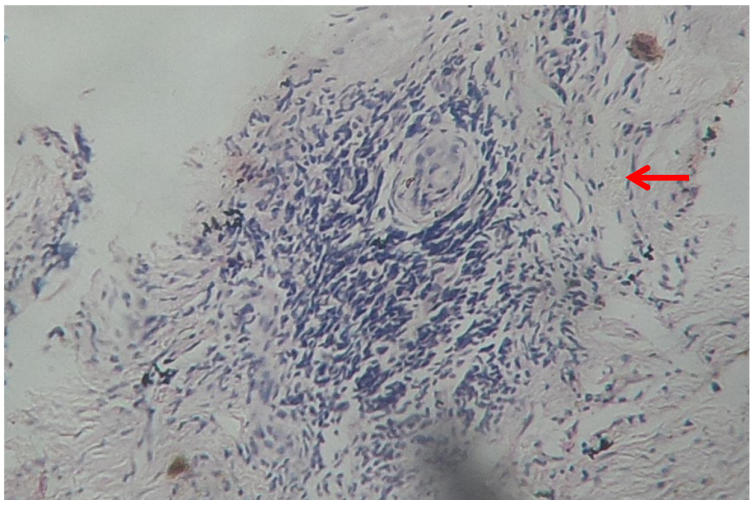
FIGURE 34: CLINICAL PHOTOGRAPH- PSEUDOPELADE OF BROCQ

FIGURE 35: DERMOSCOPIC FINDINGS IN PSEUDOPELADE OF BROCQ



Ivory white scarred areas(blue arrow) with sourrounding areas of mottled pigmentation

FIGURE 36: HISTOPATHOLOGY IN PSEUDOPELADE OF BROCQ



Dense perifollicular mononuclear inflammatory infiltrate(red arrow)

FIGURE 37: CLINICAL PHOTOGRAPH- FOLLICULITIS DECALVANS

FIGURE 38: DERMOSCOPIC FINDINGS IN FOLLICULITIS DECALVANS

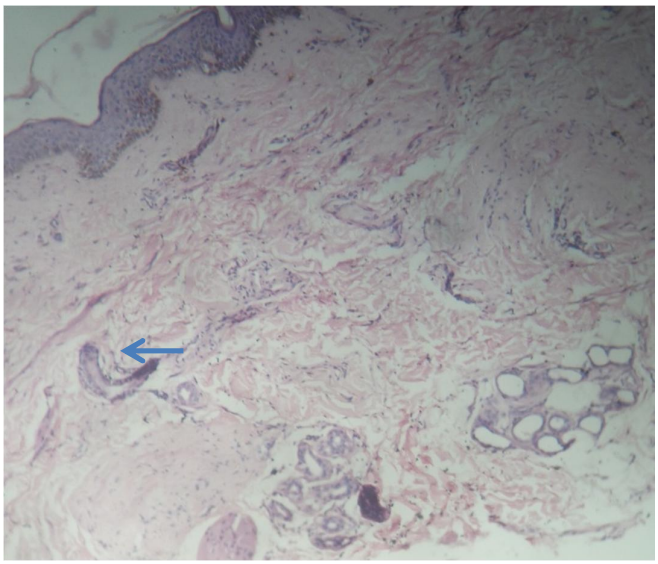
Follicular pustules in various stages of development

FIGURE 39: CLINICAL PHOTOGRAPH- FRONTAL FIBROSING ALOPECIA

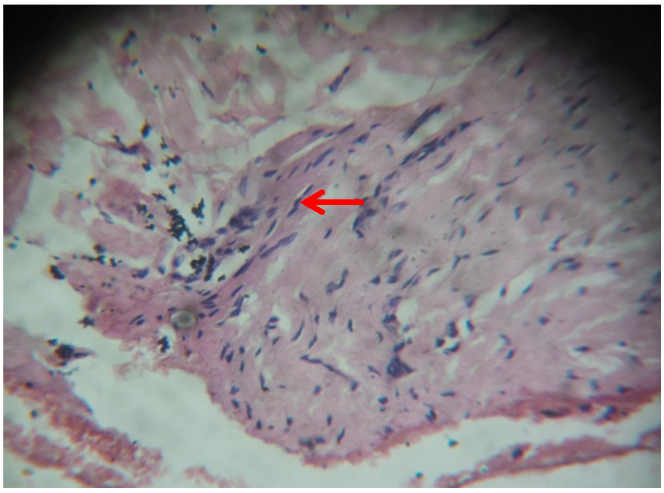
FIGURE 40: DERMOSCOPIC FINDINGS IN FRONTAL FIBROSING ALOPECIA

Loss of follicular orifices in a cream background

FIGURE 41: HISTOPATHOLOGY IN FRONTAL FIBROSING ALOPECIA



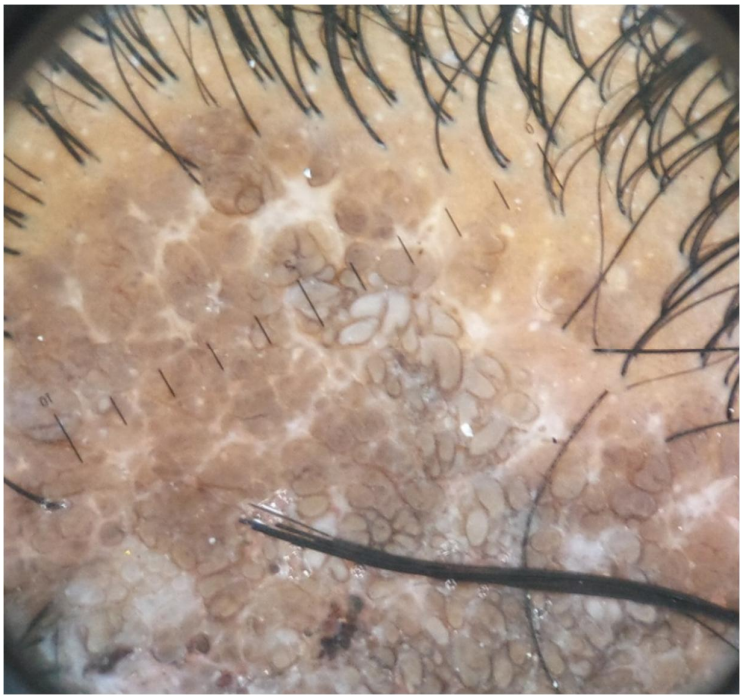
A- Low power view- Atrophic hair follicles(blue arrow) and mononuclear periappendageal inflammatory infiltrate



B- High power view- Arrector pilorus muscle with infiltration by fibroblasts (red arrow)

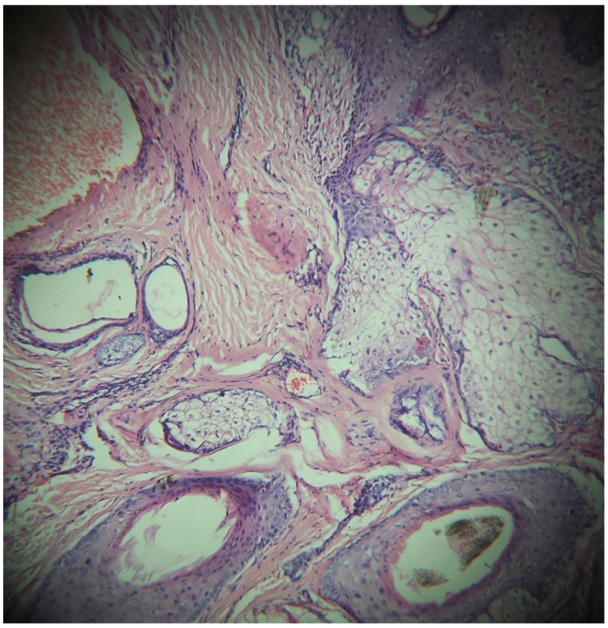
FIGURE 42: CLINICAL PHOTOGRAPH- NEVUS SEBACEOUS

FIGURE 43: DERMOSCOPIC FINDINGS IN NEVUS SEBACEOUS



Absence of hair with presence of lipid globules

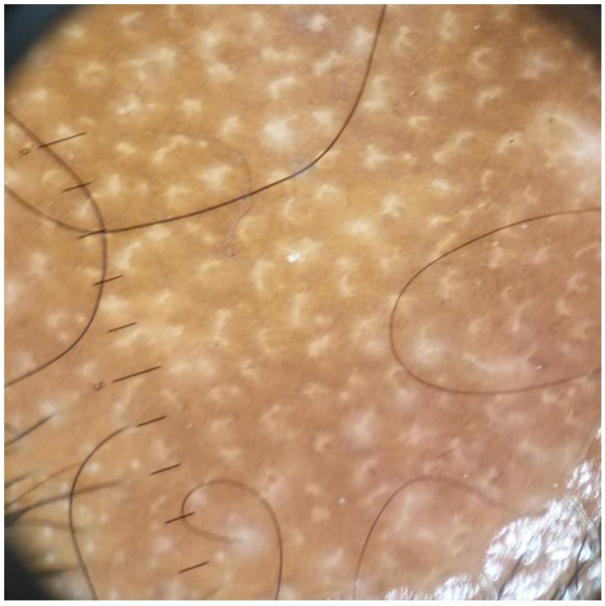
FIGURE 44: HISTOPATHOLOGY IN NEVUS SEBACEOUS



Absence of hair follicles with large sebaceous glands and apocrine glands in dermis

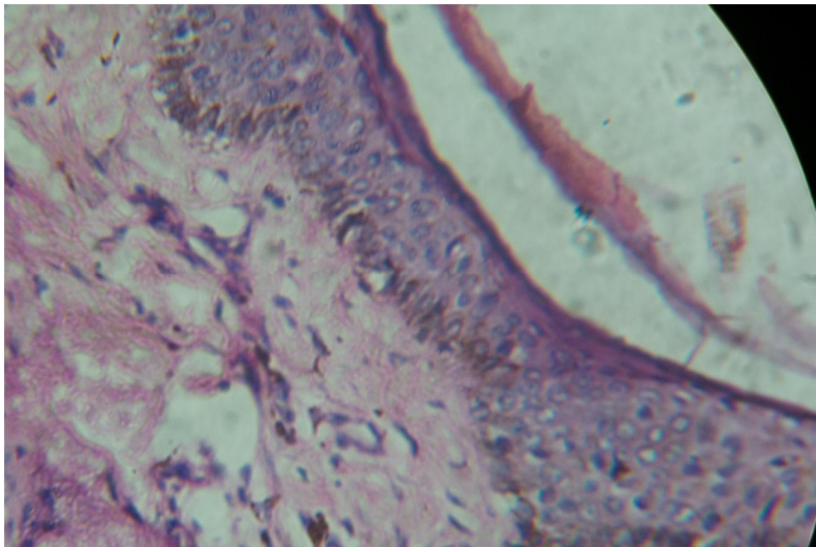
FIGURE 45: CLINICAL PHOTOGRAPH- APLASIA CUTIS

FIGURE 46: DERMOSCOPIC FINDINGS IN APLASIA CUTIS SHOWING FOLLICULAR SCARS



Stellate follicular scars

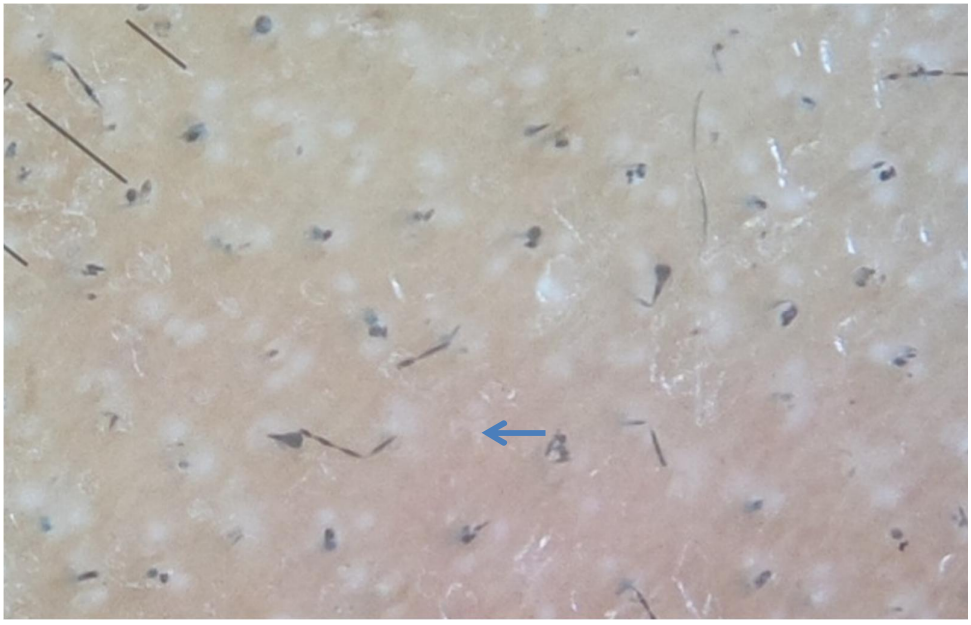
FIGURE 47: HISTOPATHOLOGY IN APLASIA CUTIS



Flattened epidermis with disorganised connective tissue

FIGURE 48: CLINICAL PHOTOGRAPH: PATIENT WITH MONILETHRIX

FIGURE 49: DERMOSCOPIC FINDINGS IN MONILETHRIX



Nodal dialations with intermodal constrictions at which there is shaft breakage

Discussion

DISCUSSION

The commonest type of alopecia in our study was alopecia areata followed by tinea capitis, androgenetic alopecia and telogen effluvium. The commonest scarring alopecia were discoid lupus eythematosus and lichen planopilaris. The commonest cause of congenital alopecia was nevus sebaceous.

ALOPECIA AREATA

Alopecia areata was the most common type of alopecia in this study accounting for nearly 25% of the total cases. The number of male patients outweighed the number of female patients. Patchy type was the commonest type (80%) followed by three patients with extensive alopecia areata (one patient each of alopecia totalis, alopecia subtotalis and diffuse alopecia areta) and one patient with ophiasis type of alopecia areata. Mane et al also had patchy alopecia areata as the commonest type of alopecia in their study (87.1%).[62]

Dermoscopically, all patients had non-scarring alopecia. Black dots (85%) and broken hair (65%) were found to be the commonest findings. This was followed by empty follicles (65%), yellow dots (60%), exclamation mark hair (55%), vellus hair (55%) and coudability sign (40%). Mane et al found yellow dots (81.8%), followed by black dots (66.6%), broken hairs (55.4%),

short vellus hair (40.9%) and tapering hairs (12.1%) as their common dermoscopic findings. Thus, yellow dots were the commonest finding in their study whereas black dots was the commonest finding in our study. The percentage of patients with black dots, broken hair, dermoscopic exclamation hair, vellus hair and dermoscopic coudablity sign are higher in our study.[61]Jain et al have reported less incidence of yellow dots in dark skinned people compared to their European counterparts consistent with findings in our study.[62.] Inui et al found black dots(44.3%), eclamation mark hair (31.7%), broken hair (45.7%), short vellus hair (44.3 %), either short vellus hair or yellow dots (94%) as their common dermoscopic findings. However, he considered black dots, broken hair and exclamation mark hair to be pathognomic for alopecia areata. These findings are similar to our study.[23]

Dermoscopic coudability sign and exclamation mark hair were common than clinical coudability sign and exclamation mark hair in occurance in our study.

Black dots, broken hair and yellow dots being the commonest findings in all the subtypes of alopecia areata in the study. This is consistent with Mane et al study.[61]

Scalp scraping and hair root examination for fungal elements were negative in all cases.

TINEA CAPITIS

It was the second most common type of alopecia in the study. Male patients below the age of thirteen years comprised the maximum proportion in the study group. Half of the patients had non-inflammatory type of tinea capitis of which maximum were of the grey patch type. The other half had inflammatory type of tinea capitis of which kerion and pustular types were the common types.

Dermoscopically, the commonest finding in cases of tinea capitis was broken hairs (100%) and black dots (92.86%). This was followed by specific findings of comma hair (71.43%), coiled hair (42.86%), cockscrew hair (42.86%).

The dermoscopic findings in non-inflammatory type consisted mainly of black dots, broken hair and scaling. Comma hair, coiled hair and cockscrew hairs were not seen in the glabrous type. This is consistent with Jain et al review of dermoscopic findings in non-inflammatory tinea capitis which had comma hair, black dots and scaling as their common findings. However, our study has found cockscrew hairs and coiled hair as other common and specific findings.[62]

Dermoscopic findings found in case of inflammatory tinea capitis were black dots, broken hair, comma hair, follicular pustules and crusting to be

present in most cases. Cockscrew hair and coiled hair were commonly seen in Pustular type of tinea capitis. Jain et al found blotchy pigmentation, comma hair, pustules and crusting as the common dermoscopic findings in inflammatory tinea capitis. However, our study found cockscrew hair and coiled hair as other common and specific findings in inflammatory tinea capitis. Black dot was a common but non-specific finding in our study in cases of inflammatory tinea capitis as it was also found in alopecia areata and trichotillomania.[62]

Scalp scraping and hair root examination in all cases showed fungal elements in form of refractile spores in and around the hair follicle.

ANDROGENETIC ALOPECIA

Androgenetic alopecia was the third most common type of alopecia in the study with almost equal sex distribution between age of 20 and 40 years.

Dermoscopic findings in androgenetic alopecia included hair shaft diameter variation (>20%), vellus hair and hair follicles with single hair follicle (>35%) in all cases (100%). Empty follicles were seen in most patients (90%) with peripilar halo being the other finding (60%). Yellow dots were seen only in one patient (10%). The findings were noted in the frontal

region and absent in the occipital region. Both male and female patients showed similar findings except for the fact that the one patient who showed yellow dots was a female patient. Kalidayalan et al in their review of dermoscopic findings in androgenetic alopecia found peripilar halo sign as the commonest finding in Asians followed by hair shaft diameter variation (>20%), presence of vellus hairs and yellow dots as the other common findings. In our study, hair follicles with a single hair (>35%) was a consistent finding with peripilar halo as the fourth commonest finding.[64] Rakowska et al designed a criteria for videodermoscopy for female androgenetic alopecia with presence of yellow dots in frontal region, lower average thickness of hair in frontal region and presence of more than 10% vellus hair in frontal region as their major criteria and increased frontal:occipital ratio of single hair pilosebaceous units, vellus hair and peripilar halo sign as their minor criteria. They found it to be 98% specific for diagnosing female androgenetic alopecia provided two major or one major and two minor criteria are fulfille.[64] Findings in our study done by hand-held dermoscopy are similar except for the less common occurance of yellow dots in our study compared to that conducted by Rakowska et al.

TELOGEN EFFLUVIUM

This was the fourth most common type of non-scarring alopecia with female sex preponderance with patients between 20 and 34 years of age.

Dermoscopy in telogen effluvium showed hair follicles with single hair (>35%) were seen in all cases (100%). Hair shaft diameter variation (<20%) (57%) and empty follicles (57%) were the other common findings.

Hair shaft diameter variation(<20%), absence of peripilar halo and presence of uniform dermoscopic findings almost all over the scalp for differentiating telogen effluvium from androgenetic alopecia. This was consistent with the criteria laid down by Rakowska et al in their study to differentiate androgentic alopecia from telogen effluvium.[64]

TRICHOTILOMANIA

Trichotillomania was the fifth most common type of non-scarring alopecia in the study with majority of the patients being females in the paediatric age group.

Black dots was the most common finding seen in all patients followed by specific findings of broken hair of varying size (83.33%) and coiled hair (50%). Perifollicular haemorrhage and V-Sign were seen in one case each. (16.67%). Abraham et al found black dots, broken hair , frayed hair, coiled

hair, empty follicular ostia and yellow dots as their common dermoscopic findings with presence of broken hair at different intervals and coiled hair and absence of exclamation mark hair as the finding for differentiating from alopecia areata. Thus, findings in our study were consistent with their findings.[63]

Scalp Scraping & hair root examination for fungal elements in all patients was negative.

ANAGEN EFFLUVIUM

Two patients with anagen effluvium, both on chemotherapy, with diffuse non-scarring alopecia presented during the study period.

Dermoscopic examination showed short terminal hair, abruptly constricted hair, numerous empty follicles and hair shaft breakage in both cases (100%).

Rakowska et al have found broken hair, monilethrix-like hair, abruptly constricted hair, hair shaft breakage, yellow dots and regrowing pig tail hairs as the common dermoscopic findings in anagen effluvium. Our study had no patients with regrowing pig tail hairs or yellow dots.[65]

LICHEN PLANOPILARIS

Lichen planopilaris was the most common scarring alopecia in the study along with discoid lupus erythematosus. There was a slight female predisposition with patients between the age of 20 and 50 years. Few patients showed cutaneous or mucosal features of lichen planus.

Dermoscopically, all patients showed reduction in hair follicles and white dots. Perifollicular violaceous pigmentation and dystrophic hair were the other common finding (57.14%). Wickham's striae, empty follicles, follicular plugs and follicular casts were seen in 1 patient each (14.29%).

Jain et al found perifollicular casts, perifollicular blue-grey pigmentation and white dots as the common dermoscopic findings in lichen planopilaris. Although white dots were a universal and perifollicular pigmentation were a constant finding in our study, follicular cast was a rare finding. Wickham striae was reported in one patient in our study and dystrophic hair was a common finding.[62]

Biopsy and histopathologic examination were done in 6 out 7 patients and were consistent with dermoscopic diagnosis of lichen planopilaris.

DISCOID LUPUS ERYTHEMATOSUS

In our study, discoid lupus erythematosus and lichen planopilaris were found to be the commonest scarring alopecia. It showed a strong female predisposition with all patients between the age of 20 to 40 years. Few patients complained of photosensitivity and showed discoid cutaneous lesions.

Dermoscopy showed reduced number of hair follicles in all patients (100%). Follicular plugs(85.71%) was the commonest finding followed by interfollicular pigmentation (71.43%). Telengectasia (57.14%), was the other common finding. Lanuti et al reported reduced hair follicles, follicular keratotic plugs, blue grey pigmentation, arborizing vessels, white atrophic patches, follicular red dots and scaling as common dermoscopic features. Although our study had similar findings to the study by Lanuti et al, none of our patients had follicular red dots.[66]

Hisopathological examination was done in 6 out of 7 patients. Biopsy findings were consistent with discoid lupus erythematosus in all cases.

PSEUDOPELADE OF BROCQ

Two female patients in the age group between 35 and 40 years presented with Pseudopelade of Brocq.

Dermoscopic examination showed reduction in hair follicles in both patients with perifollicular brown pigmentation, ivory white areas, dystrophic hair and empty follicles as the other findings. These were consistent with Jain et al who found reduced hair follicles, hypopigmented scarred areas and dystrophic hair as the main dermoscopic findings in their study.[62]

Histopathological examination was consistent with Pseudopelade of Brocq.

FOLLICULITIS DECALVANS

2 patients with folliculitis decalvans with follicular pustules and scarring alopecia presented during the study period.

Dermoscopy showed follicular pustules in various stages of development with reduced hair follicles in the surrounding regions. Jain et al found tufting of hair follicles, twisted capillary loops, follicular pustules and loss of hair follicles as their principal dermoscopic findings in folliculitis decalvans. None of our patients exhibited obvious tufting or twisted capillary loops on dermoscopic examination.[62]

Histopathologic examination showed non-specific findings in both cases.

FRONTAL FIBROSING ALOPECIA

A single female patient with scarring frontal fibrosing alopecia was seen during study period.

Dermoscopic examination showed reduced hair follicles over a cream coloured background with multiple hair follicles with single hair. A single hair with follicular cast was seen.

Biopsy from the area and histopathologic examination was consistent with frontal fibrosing alopecia.

NEVUS SEBACEOUS

2 male patients with clinical diagnosis of nevus sebaceous presented during the study period.

Dermoscopic examination showed absence of hair follicles with widespread lipid globules.

Histopathologic examination was consistent with nevus sebaceous.

APLASIA CUTIS

A one year male patient with patchy scarring alopecia since birth clinically diagnosed as aplasia cutis presented during study period.

Dermoscopy showed absence of hair over the patch with multiple stellate scars over the patch.

Histopathologic examination was consistent with aplasia cutis.

MONILETHRIX

A 2 month old baby with Holt-Oram syndrome presented during study period with absence of hair over scalp and eyebrows since birth.

Dermoscopic examination of scalp and eyebrows showed hair shafts with monilethrix.

Conclusion

CONCLUSION

- Non-scarring alopecia was more common than scarring alopecia in our study. The commonest non-scarrring alopecia was alopecia areata followed by tinea capitis and androgenetic alopecia. The commonest scarring alopecia were discoid lupus erythematosus and lichen planopilaris. The commonest congenital alopecia was nevus sebaceous.
- Alopecia areata: Patchy type of alopecia areata was commonest in our study. The common dermoscopic findings black dots and broken hair, followed by yellow dots, exclamation mark hair, vellus hair and coudability sign. The findings were almost similar in all types of alopecia areata.
- Tinea capitis: Equal incidence of inflammatory and non-inflammatory tinea capitis was found in our study. The common dermoscopic findings included black dots, broken hair, comma hair, coiled hair and cockscrew hair. Scaling was seen in non-inflammatory tinea capitis and follicular pustles and crusting were seen in inflammatory tinea capitis. Thus, our study finds coiled and cockscrew hair as specific common findings in tinea capitis in association with already reported comma hair. All cases showed positive scalp scraping and hair root examination for fungal elements.

- Androgenetic alopecia: Dermoscopic examination in both male and female patients revealed hair shaft diameter variation (>20%), vellus hair, hair follicles with single hair (>35%) and peripilar halo as significant and common findings with significant difference in fronto-occipital dermoscopic findings in all patients.
- Telogen effluvium: It closely mimicked androgenetic alopecia clinically but was differentiated dermoscopically by absence of significant hair shaft diameter variation, absence of significant number of vellus hair & absence of peripilar halo sign. The consistent finding in telogen effluvium in our study was multiple (>35%) hair follicles with single hair. There was no significant difference in fronto-occipital dermoscopic findings in all patients.
- Trichotillomania: Broken hair of varying lengths and coliled hair were common specific findings. Black dots was common non-specific finding.
- Anagen effluvium: Short terminal hair, abruptly constricted hair and hair shaft breakage were consistent dermoscopic findings.
- Lichen Planopilaris: White dots with reduced hair density were present dermoscopically in all patients. The other common findings were perifollicular violaceous pigmentation and dystrophic hair.

- Discoid lupus erythematosus: Reduced hair follicles, follicular plugs, interfollicular pigmentation and telengectasia were the common dermoscopic findings.
- Frontal fibrosing alopecia: Reduced hair follicles over a cream background were seen dermoscopically in a single patient of this condition.
- Pseudopelade of Brocq: Reduction in hair follicles with perifollicular brown pigmentation, ivory white areas and dystrophic hair were found dermoscopically in our study.
- Folliculitis decalvans: Follicular pustules in different stages of evolution in a background of scarring alopecia were seen dermoscopically in both patients of this condition in our study.
- Aplasia cutis: Stellate scars with absent hair follicles was seen in the single patient of this condition in our study.
- Nevus sebaceous: Lipid globules in a background of absent hair follicles was found in both patients of nevus sebaceous dermoscopically in our study.
- Monilethrix: Single hair shaft disorder seen in the study was monilethrix.

Bibliography

REFERENCES

1. Wadhwa S.L., Khopkar U, Nischal K.C. Hair and Scalp disorders. IADVL Textbook of Dermatology. 3 e;2008; 864-949.
2. Benvenuto-Andrade C et al. Differences between polarized light dermoscopy and immersion contact dermoscopy for the evaluation of skin lesions. Arch Dermatol. 2007; 143(3):329-38
3. Inui S, Nakajima T, Itami S. Dry dermoscopy in clinical treatment of alopecia areata. J Dermatol. 2007; 34(9): 635-9
4. Michael D. Loffreda. Lever's Histopathology of the skin. Inflammatory Diseases of Hair Follicles, Sweat Glands and Cartilage. Wolters Kluwer publications. 2010; 470-473
5. Rudnicka L et al. Trichoscopic structures and patterns. Atlas of trichoscopy. Springer publications; 2012; 11-110
6. Djabali K et al. Recurrent missense mutations in hair keratin gene hHb6 in monilethrix. Clin Exp Dermatol. 2003; 28(2):206-10
7. Mirmirani P, Huang KP, Price VH. A practical algorithmic approach to diagnose hair shaft disorders. Int J Dermatol. 2011; 50(1):1-12
8. Rakowska A, Slowinska M, Czuwara J, Rudnicka L. Dermoscopy as a tool for rapid diagnosis of monilethrix. J Drugs Dermatol. 2007; 6(2):222-4

9. Zitelli JA.Pseudomonilethrix. An artifact. Arch dermatol. 1986; 122(6)688-90
10. Rakowska A, Kowalska-Oledzka E, Slowinska M, Rosinska D, Rudnicka L. Hair shaft videodermoscopy in Netherton syndrome. Pediatr dermatol. 2009;26(3):320-2
11. Bartels NG, Blume-Peytavi U. Hair loss in children. Hair growth and disorders. Leipzig: Springer; 2008. P. 293-4
12. Chernosky ME, Owens DW. Trichorrhexis nodosa. Clinical and investigative studies. Arch Dermatol. 1966; 94(5):577-85
13. Whiting DA, DY Lc. Office diagnosis of hair shaft defects. Semin Cutan Med Surg. 2006; 25(1):24-34
14. Rakowska A, Slowinska M, Kowalska-Oledzka E, Rudnicka L. Trichoscopy in genetic hair shaft abnormalities. J Dermatol Case Rep. 2008(2):14-20
15. Cheng AS, Bayliss SJ. The genetics of hair shaft disorders. J Am Acad Dermatol. 2008; 59(1):1-22; quiz 3-6
16. Zhou X, Khan SG, Tamura D, Patronas NJ, Zein WM, Brooks BP. Brittle hair, developmental delay, neurologic abnormalities and photosensitivity in a 4 year old girl. J Am Acad Dermatol. 2010; 63(2):323-8

17. Itin PH, Sarasin A, Pittelkow MR. Trichothiodystrophy: update on the sulphur-deficient brittle hair syndromes. J Am Acad Dermatol. 2001; 44(6):891-920; quiz 1-4
18. Bennassar A, Ferrando J, Grimalt R. Congenital atrichia and hypotrichosis. World J Pediatr. 2011; 7(2):111-7
19. Mehta U, Brunworth J, Fete TJ, Sindwani R. Head and neck manifestations and quality of life of patients of ectodermal dysplasia . Otolaryngol Head Neck Surg. 2007; 136(5):843-7
20. Zadruska M, Rakowska A, Rudnicka L. Hair abnormalities in patients of ectodermal dysplasia: clinical evaluation, trichogram and trichoscopy.
21. Kolalapudi Anjaneyulu Seetharam. Alopecia Areata: An update. Indian Journal of Dermatology, Venereology and Leprosy. September-October 2013; Vol 79; Issue 5
22. Rudnicka et al. Alopecia Areata: Atlas of trichoscopy.Springer publications. 2012; 206-220
23. Inui S, Nakajima T, Nakagawa K, Itami S. Clinical significance of dermoscopy in alopecia areata: analysis of 300 cases. Int J Dermatol. 2008; 47(7): 688-693.
24. Rudnicka L, Olsewzska M, Rakowska A. Trichoscopy: a new method for diagnosing hair loss. J Drugs Dermatol. 2008; 7(7):651-4.

25. Otberg N, Finner AM, Shapiro J. Androgenetic alopecia. Endocrinol Metab Clin North Am. 2007; 36(2):379-98
26. Inui S, Nakajima T, Itami S. Scalp dermoscopy of androgenetic alopecia in Asian people. J dermatol. 2009; 36(2):82-5
27. Rakowska A, Slowinska M, Kowalska- Oledzka E, Rudnicka L. Dermoscopy in female androgenetic alopecia. Int J Trichol. 2009 1(2):123-130.
28. Karadag Kose O, Gulec AT. Clinical evaluation of alopecias using hand held dermatoscope. J Am Acad Dermatol. 2011; 67:206-14
29. Michael D. Loffreda. Lever's Histopathology of the skin. Inflammatory Diseases of Hair Follicles, Sweat Glands and Cartilage. Wolters Kluwer publications. 2010; 459-503
30. Trueb RM. Systematic approach to hair loss in women. J Dtsch Dermatol Ges. 2010; 8(4): 284-297,98
31. Slowinska M. The value of videodermoscopy in differential diagnosis of androgenetic alopecia. Warsaw: Medical university of Warsaw; 2010
32. Dhurat RP, Deshpande DJ. Loose Anagen hair syndrome. Int J Trichol. 2010; 2(2):96-100
33. Antaya RJ, Sideridou E, Olsen EA. Short Anagen Syndrome. J Am Acad Dermatol. 2005; 53:S130-4

34. Grimalt R. Practical approach to scalp disorders. J Investig Dermatol Symp Proc. 2007; 12(2):10-4

35. Gupta AK, Summerbell RC. Tinea Capitis. Med Mycol. 2000; 38(4):255-87

36. Sarabi K, Khachemoune A. Tinea capitis: a review. Dermatol Nurs. 2007; 19(6):525-9; quiz 30

37. Sandoval AB, Oritz JA et al. Dermoscopic pattern in Tinea capitis. Rev Iberoam Micol. 2010; 27(3):151-2

38. Lochner C et al. The validity of DSM IV TR criteria B and C of hair pulling disorder (trichotillomania): evidence from clinical study. Psychiatry Res. 2011; 189(2):276-280

39. Sah DE, Koo J, Price VH. Trichotillomania. Dermatol Ther.2008; 21(1):13-21

40. Lee DY, Lee JH, Yang JM, Lee ES. The use of dermoscopy for the diagnosis of trichotillomania. J Eur Acad Dermatol Venerol. 2009; 23(6):731-2

41. Lee DY, Lee JH, Yang JM, Lee ES. The use of dermoscopy for the diagnosis of trichotillomania. J Eur Acad Dermatol Venerol. 2009; 23(6):734-6

42. Michael D. Loffreda. Lever's Histopathology of the skin. Inflammatory Diseases of Hair Follicles, Sweat Glands and Cartilage. Wolters Kluwer publications. 2010; 459-503
43. Hantash BM, Schwartz RA. Traction alopecia in children. Cutis. 2003; 71(1):18-20
44. Price V, Mirmirani P. Cicatricial Alopecia: An approach to diagnosis and management. New York: Springer,2011
45. Rakowska A, Slowinska M, Kowalska- Oledzka E, Rudnicka L. Trichoscopy in cicatrical alopecia J Drugs Dermatol. 2012; 11:753-8.
46. Duque-Estrada B, Tamler C, Sodre CT, Barcaui CB. Dermoscopy patterns of cicatricial alopecia resulting from discoid lupus erythematosus and lichen planopilaris. An Bras Dermatol. 2010;85(2):179-183.
47. Abraham LS, Piniero-Maceria J, Duque-Estrada B, Barcaui CB, Sodre CT. Pin point white in scalp: Dermoscopic and histopathological correlation. J Am Acad Dermatol. 2010; 63(4):721-2
48. Narciss Mobini, Sonia Toussaint, Hideko Kamino. Lever's Histopathology of the skin. Noninfectious erythematous, papular and squamous diseases. Wolters Kluwer publications. 2010; 169-205.

49. Chew AL, Bashir SJ, Wain EM, Fenton D, Stefanato CM. Expanding the spectrum of frontal fibrosing alopecia: A unifying concept. J Am Acad Dermatol. 2010; 63(4):653-60
50. Tan KT, Messenger AG. Frontal fibrosing alopecia : clinical presentations and prognosis. Br J Dermatol. 2009; 160(1):75-9
51. Inui S, Nakajima T, Shono F, Itami S. Dermoscopic findings in frontal fibrosing alopecia: report of four cases. Int J Dermatol. 2008; 47(8):796-9
52. Rudnicka L., Olszewska M, Rakowska A, Slowinska M. Trichoscopy update 2011. J Dermatol case rep. 2011; 5(4):82-8
53. Walling HW, Sontheimer RD. Cutaneous lupus erythematosus: issues in diagnosis & treatment. Am J Clin Dermatol. 2009; 10(6):365-81
54. Harry Winfield, Christine Jawrowsky . Lever's Histopathology of the skin. Connective tissue disorders. Wolters Kluwer publications. 2010; 279-310.
55. Somani N, Bergfeld WF. Cicatricial alopecia: classification and histopathology. Dermatol Ther. 2008; 21(4):221-37
56. Elston DM. Tufted folliculitis. J Cutan Pathol. 2011; 38(7):595-6
57. Otberg N, Kang H, Alzolibani AA. Folliculitis decalvans. Dermatol Ther. 2008; 21(4):238-44

58. Wu WY, Otberg N, McElwee KJ, Shapiro J. Diagnosis and management of primary cicatricial alopecia: Part II. Skinmed. 2008;7(2)78-83

59. Whiting DA. Cicatricial alopecia: clinico-pathological findings and treatment.Clin Dermatol 2001; 19: 211–25.

60. Alzolibani AA, Kang H, Otberg N, Shapiro J. Pseudopelade of Brocq. Dermatol Ther. 2008; 21(4):257-63

61. Mane M, Nath AK, Thappa DM. Utility of dermoscopy in alopecia areata. Indian J Dermatol 2011 56:407-11

62. Jain N, Doshi B, Khopkar U. Trichoscopy in Alopecias: Diagnosis Simplified. International J Trichol, 2013; 5(4)

63. Abraham LS, Torres FN, Luna A. Dermoscopic clues to distinguish trichotillomania from patchy alopecia areata;. An Bras Dermatol. 2010;85(5):723-6

64. Kaliyadan F, Nambiar A, Vijayaraghavan S. Androgenetic alopecia: An update. Indian J Dermatol Venereol Leprol 2013;79:613-25

65. Rudnicka L et al. Anagen Effluvium. Atlas of trichoscopy. Springer publications; 2012; 246-254

66. Lanuti E, Miteva M, Romanelli P, Tosti A. Trichoscopy and histopathology of follicular keratotic plugs in scalp discoid lupus erythematosus. Int J Trichol 2012; 4:36-8

Annexures

PATIENT CONSENT FORM

Title of the study: Clinical study of dermoscopic findings in non-cicatricial & cicatricial alopecia

Name of the Participant:

Name of the Principal investigator: Dr.Vivek Shah

Name of the Institution: Rajiv Gandhi Government General Hospital,Chennai

Documentation of the informed consent

I _____ have read the information in this form (or it has been read for me). I was free to ask any questions and they have been answered. I am over 18 years of age and exercising my free power of choice, hereby give my consent to be included as a participant in the study.

1. I have read and understood this consent form and the information provided to me.
2. I have had the consent document explained to me.
3. I have been explained about the nature of the study.

4. My rights and responsibilities have been explained to me by the investigator.

5. I have informed the investigator of all the treatments I am taking or have taken in the past.

6. I agree to cooperate with the investigator and I will inform her immediately if I suffer unusual symptoms.

7. I have not participated in any research study at any time .

8. I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.

9. I hereby give permission to the investigators to release the information obtained from me as a result of participation in this study to the sponsors ,regulatory authorities, Governmentt agencies and IEC.I understand that they are publicly presented.

10. My identity will be kept confidential if my data are publicly presented.
11. I have had my questions answered to my satisfaction.
12. I have decided to be in the research study

13. I am aware that if I have any question during this study, I should contact at one of the addresses listed above. By signing this consent form I attest that the information given in this document has been clearly explained to me and apparently understood by me. I will be given a copy of this consent document.

Participant's initials:_____

For adult participants:

Name and signature/thumb impression of the participant(or legal representative if participant incompetent)

_____ _____ _____

Name Signature Date

Name and signature of impartial witness (required for illiterate patients):

_____ _____ _____

Name Signature Date

Address and contact number of the impartial witness:

Name and Signature of the investigator or his representative obtaining consent:

_____ _____ _____

Name Signature Date

For Children being enrolled in research:
Whether child's assent was asked: Yes / No (Tick one)

[If the answer to be above question is yes :

You agree with the manner in which assent was asked for from your child and given by your child. You agree to have your child take part in this study].

[If answer to be above question No, give reason (s)

Although your child did not or could not give his or her assent, you agree to your child's participation in this study.

Name and Signature of / thumb impression of the participant's parent(s) (or legal representative)

Name _____ Signature_____ Date_____

Name _____ Signature_____ Date_____

Name and Signature of impartial witness (required for parents of participant child illiterate):

Name _____ Signature_____ Date_____

Address and contact number of the impartial witness: _____

PROFORMA

S. No.: Date:

NAME: AGE: SEX:M/F

OCCUPATION:

ADDRESS:

COMPLAINTS:

OTHER RELEVANT HISTORY:

TREATMENT HISTORY:

CLINICAL EXAMINATION:

CLINICAL PHOTOGRAPH NO.:

DERMOSCOPIC FINDINGS:

Hair density:

Hair shaft diameter variation:

Hair shaft defects:

Yellow dots: Black dots: White dots:

Follicular orifices:

Perifollicular halo:

Scaling:

Atrophy:

Telengectasia:

Capillary loops:

Erythema:

Scalp pigmentation:

Exclamation mark hair:

Broken hairs:

Vellus hair:

Depigmented hair:

Any other findings:

DERMOSCOPIC PHOTOGRAPH NO.:

OTHER INVESTIGATIONS:

Master Chart

MASTER CHART

ALOPECIA AREATA

Sl. No.	Age	Sex	Occupation	Clinical type	Type of disease	Hair pull test	Coudablity sign	Exclamation mark hair	Hair density	Hair follicles	Black dots	Yellow dots	Broken hair	Dermoscopic Exclamation mark hair	Vellus hair	Empty follicle	Deramscopic coudablity	V-Sign	Scraping for fungus
1	10	M	-	NS	Ophiasis	-	-	-	Reduced	+	+	+	+	-	-	+	-	-	-
2	16	M	Student	NS	Patchy	+	-	-	Reduced	+	+	+	+	+	-	+	-	-	-
3	32	M	Medical representative	NS	Patchy	+	+	-	Reduced	+	+	+	+	-	-	+	+	-	-
4	27	M	Village administrative officer	NS	Patchy	-	-	-	Reduced	+	+	-	-	-	+	+	+	-	-
5	25	M	Salesman	NS	Patchy	-	-	-	Reduced	+	+	-	-	-	+	-	-	-	-
6	28	M	Trader	NS	Patchy	+	+	-	Reduced	+	+	-	+	-	-	+	+	-	-
7	32	M	Gym instructor	NS	Alopecia totalis	-	-	+	Reduced	+	+	+	+	+	-	+	-	-	-
8	9	F	Student	NS	Patchy	+	+	-	Reduced	+	+	+	+	+	-	+	+	-	-
9	40	F	Housewife	NS	Patchy	-	-	-	Reduced	+	-	-	-	+	+	+	-	-	-
10	20	F	Unemployed	NS	Diffuse	-	-	-	Reduced	+	+	+	+	+	-	+	-	-	-
11	45	M	Banker	NS	Patchy	+	+	-	Reduced	+	+	+	-	-	+	-	-	+	-
12	28	M	Labourer	NS	Patchy	+	-	+	Reduced	+	-	-	-	+	+	+	+	-	-
13	28	F	Housewife	NS	Patchy	+	+	+	Reduced	+	+	+	+	+	-	-	+	-	-

Sl. No.	Age	Sex	Occupation	Clinical type	Type of disease	Hair pull test	Coudablity sign	Exclamation mark hair	Hair density	Hair follicles	Black dots	Yellow dots	Broken hair	Dermoscopic Exclamation mark hair	Vellus hair	Empty follicle	Deramscopic coudablity	V-Sign	Scraping for fungus
14	29	F	Housewife	NS	Alopecia subtotalis	+	-	-	Reduced	+	-	+	-	-	+	+	-	-	-
15	8	M	Student	NS	Patchy	-	-	-	Reduced	+	+	+	+	-	+	+	-	-	-
16	6	F	-	NS	Patchy	+	-	-	Reduced	+	+	-	+	+	+	-	-	-	-
17	28	F	Housewife	NS	Patchy	+	-	-	Reduced	+	+	-	-	-	-	-	-	-	-
18	12	F	Student	NS	Patchy	+	+	-	Reduced	+	+	-	+	+	+	+	+	-	-
19	18	F	Student	NS	Patchy	+	-	-	Reduced	+	+	+	+	+	+	-	-	-	-
20	9	M	Student	NS	Patchy	+	+	-	Reduced	+	+	+	+	+	+	-	+	-	-

TINEA CAPITIS

Sl. No.	Age	Sex	Occupation	Clinical type	Type of tinea capitis	Hair pull test	Coudability sign	Hair density	Hair follicle	Black dots	Broken hair	Comma hair	Coiled hair	Cockscrew hair	Follicular pustules	Crusting	Scaling	Zig-zag hair	Scraping for fungus
1	8	M	Student	NS	Pustular	+	-	Reduced	+	+	+	+	+	+	+	+	-	+	+
2	23	M	Labourer	NS	Glabrous	-	-	Reduced	+	+	+	-	-	-	-	-	+	-	+
3	50	F	Housewife	NS	Glabrous	-	-	Reduced	+	+	+	-	-	-	-	-	+	-	+
4	6	F	-	NS	Kerion	+	-	Reduced	Obscured over lesion	+	+	+	+	+	+	+	-	-	+
5	4	M	-	NS	Pustular	+	-	Reduced	+	+	+	+	+	+	+	+	-	-	+
6	5	M	-	NS	Grey patch	+	-	Reduced	+	+	+	+	-	-	-	-	+	-	+
7	5	F	-	NS	Grey patch	+	-	Reduced	+	+	+	+	+	+	-	-	+	-	+
8	14	M	Student	NS	Kerion	+	-	Reduced	Obscured over lesion	+	+	-	-	-	+	+	-	-	+
9	6	M	Student	NS	Grey patch	+	-	Reduced	+	+	+	+	-	-	-	-	+	-	+
10	6	F	Student	NS	Grey patch	+	-	Reduced	+	+	+	+	+	+	-	-	+	-	+
11	8	M	Student	NS	Pustular	+	-	Reduced	+	-	+	+	+	+	+	+	-	-	+
12	2 months	M	-	NS	Incognito	-	-	Reduced	+	+	+	+	-	-	-	+	-	-	+
13	5	M	-	NS	Kerion	+	-	Reduced	Obscured over lesion	+	+	+	-	-	+	+	-	-	+
14	9	F	Student	NS	Grey patch	+	-	Reduced	+	+	+	-	-	-	-	-	+	-	+

ANDROGENETIC ALOPECIA

Sl. No.	Age	Sex	Occupation	Clinical type	Hair loss pattern	Duration	Hair density	Hair follicles	Hair shaft diameter variation	Vellus hair	Peripilar halo	Empty follicle	Hair follicles with single hair	Yellow dots	Occipital scalp dermoscopy	Fronto-occipital dermoscopic variation
1	22	M	Artist	NS	FTR	1 year	Reduced	+	+(>20%)	+	-	+	+(>35%)	-	Normal	+
2	20	M	Air force	NS	FTR TFV	2 years	Reduced	+	+(>20%)	+	-	+	+(>35%)	-	Normal	+
3	28	M	Police inspector	NS	FTR TFVT	8 months	Reduced	+	+(>20%)	+	+	+	+(>35%)	-	Normal	+
4	35	F	Housewife	NS	TF	2 years	Reduced	+	+(>20%)	+	+	+	+(>35%)	+	Normal	+
5	40	F	Housewife	NS	TF	1 year	Reduced	+	+(>20%)	+	-	-	+(>35%)	-	Normal	+
6	28	M	Labourer	NS	FTR TV	8 months	Reduced	+	+(>20%)	+	+	+	+(>35%)	-	Normal	+
7	34	M	Shopkeeper	NS	FTR TFV	1 year	Reduced	+	+(>20%)	+	+	+	+(>35%)	-	Normal	+
8	38	F	Office desk	NS	FPT	6 years	Reduced	+	+(>20%)	+	-	+	+(>35%)	-	Normal	+
9	32	M	Trader	NS	FTR TF	2 years	Reduced	+	+(>20%)	+	+	+	+(>35%)	-	Normal	+
10	26	F	Housewife	NS	TF	2 years	Reduced	+	+(>20%)	+	+	+	+(>35%)	-	Normal	+

TELOGEN EFFLUVIUM

Sl. No.	Age	Sex	Occupation	Clinical type	Hair loss pattern	Duration	Hair density	Hair follicles	Hair shaft diameter variation	Vellus hair	Peripilar halo	Empty follicle	Hair follicles with single hair	Yellow dots	Occipital scalp dermoscopy
1	22	F	Typist	NS	Frontal	1 year	Reduced	+	-	-	-	-	+(>35%)	-	Normal
2	34	F	Housewife	NS	Frontal	3 months	Reduced	+	+(<20%)	-	-	+	+(>35%)	-	Normal
3	28	M	Army officer	NS	Frontal	6 months	Reduced	+	+(<20%)	-	-	+	+(>35%)	-	Normal
4	26	F	Housewife	NS	Diffuse	1 year	Reduced	+	+(<20%)	Few	-	+	+(>35%)	-	Normal
5	31	F	Housewife	NS	Diffuse	6 months	Reduced	+	-	-	-	+	+(>35%)	-	Normal
6	28	F	Housewife	NS	Diffuse	4 months	Reduced	+	-	Few	-	-	+(>35%)	-	Normal
7	23	F	Housewife	NS	Diffuse	8 months	Reduced	+	+(<20%)	Few	-	-	+(>35%)	-	Normal

TRICHOTILLOMANIA

Sl. No.	Age	Sex	Occupation	Clinical type	Number of patches	Size	Duration	Hair pull test	Coudability sign	Hair density	Hair follicle	Black dots	Broken hair (Irregular length)	Coiled hair	Perifollicular haemorrhage	V sign	Scraping
1	4	F	-	NS	2	6*4 cm	3 months	+	-	Reduced	+	+	+	+	-	-	-
2	7	F	Student	NS	1	5*4 cm	4 months	+	-	Reduced	+	+	+	+	-	-	-
3	14	M	Student	NS	1	6*4 cm	3 months	+	-	Reduced	+	+	+	-	-	-	-
4	6	F	Student	NS	1	8*6 cm	4 months	+	-	Reduced	+	+	-	+	+	-	-
5	7	M	Student	NS	1	4*3 cm	3 months	-	-	Reduced	+	+	+	-	-	-	-
6	28	F	Housewife	NS	1	3*2 cm	2 months	+	-	Reduced	+	+	+	-	-	+	-

ANAGEN EFFLUVIUM

Sl. No.	Age	Sex	Occupation	Clinical type	Hair loss pattern	Duration	Hair pull test	Hair density	Hair follicles	Short terminal hair	Abruptly constricted hair	Perifollicular pigmentation	Empty follicle	Hair shaft breaks
1	23	M	Coolie	NS	Diffuse	15 days (Post-chemotherpay)	Positive	Reduced	+	+	+	Brown	+	+
2	68	M	Unemployed	NS	Diffuse	22 days (Post chemotherapy)	Positive	Reduced	+	+	+	-	+	+

DISCOID LUPUS ERYTHEMATOSUS

Sl. No.	Age	Sex	Occupation	Clinical type	Type of hair loss	Number of patches	Size of patches	Duration	Other skin lesions	Hair pull test	Hair density	Hair follicle	Pigmentation	Follicular plugs	Telengectasia	Perifollicular Scaling	Atrophy	Histopathology
1	34	F	Housewife	No hair follicles visualised No atrophy	Patchy	1	4*2 cm	1 month	-	+	Reduced	Obscured over lesion	Interfollicular brown	-	+	+	-	Not done
2	28	F	Labourer	Scarring	Patchy	3	4*3 to6*4 cms	8 months	Photosensitivity+	-	Reduced	Reduced	Interfollicular brown	+	-	-	-	FP, IFEA, BCDORS, PFI
3	35	F	Housewife	Scarring	Patchy	6	2*1 to 4*3 cms	9 months	-	-	Reduced	Reduced	Interfollicular brown	+	-	-	-	FP, IFEA, BCDORS, CB, PFI
4	23	F	Student	Scarring	Patchy	3	2*1 to 4*3 cms	3 months	Discoid lesions over face+	-	Reduced	Reduced	-	+	+	+	-	FP, IFEA, PFI
5	38	F	Housewife	Scarring	Patchy	1	3*2 cm	2 months	-	-	Reduced	Reduced	Interfollicular brown	+	+	-	-	FP, BCDORS, PFI
6	40	F	Housewife	Scarring	Diffuse	-	Frontoparietal area	2 years	Discoid lesions over face, extremity	-	Reduced	Reduced	-	+	-	-	+	FP, IFEA, Fibrosis in dermis
7	20	M	-	Scarring	Patchy	2	3*2 cm	6 months	Discoid lesions over face, extremity	-	Reduced	Reduced	Interfollicular brown	+	+	-	-	FP, IFEA, BCDORS, PFI

LICHEN PLANOPILARIS

Sl. No.	Age	Sex	Occupation	Clinical type	Type of hair loss	Number of patches	Size of patches	Duration	Other skin lesions	Hair pull test	Hair density	Hair follicle	White dots	Pigmentation	Empty follicles	Wickham stria e	Dystrophic hair	Follicular plugs	Follicular casts	Histopathology
1	46	M	Salesman	S	Patchy	4	4*3 cm to 10* 7 cms	2 years	Violaceous buccal mucosal pigmentation	-	Reduced	Reduced	+	Perifollicular-violaceous	-	-	+	+	-	FP, HGI, BCDORS, PFI
2	38	F	Desk Secretary	S	Patchy	4	3*2 to 4*2 cm	8 months	-	-	Reduced	Reduced	+	-	-	-	-	-	-	HGI, BCDORS, PI, PFI
3	40	F	Housewife	S	Patchy	2	2*1 to 3*2 cms	4 months	-	-	Reduced	Reduced	+	Perifollicular-violaceous	+	-	-	-	-	BCDORS, PI, PFI
4	28	M	Housewife	S	Patchy	2	2*1 to 4*3 cms	2 months	Violaceous buccal mucosal pigmentation	-	Reduced	Reduced	+	Pigment globules-brown black	-	+	+	-	-	Not done
5	45	F	Housewife	S	Patchy	1	10*7 cm	1 year	-	-	Reduced	Reduced	+	-	-	-	-	-	+	HGI, BCDORS, PI, PFI
6	34	F	Computer assisstant	S	Patchy	2	2*1 & 4*2 cms	8 months	Violaceous plaques over volar aspect of forearm & shins	-	Reduced	Reduced	+	-	-	-	+	-	-	HGI, BCDORS, PFI
7	28	M	Sales manager	S	Patchy	1	5*4 cms	1 year	-	-	Reduced	Reduced	+	Perifollicular-violaceous	-	-	+	-	-	FP, BCDORS, PFI

PSEUDOPELADE OF BROCQ

Sl. No.	Age	Sex	Occupation	Clinical type	Type of hair loss	Number of patches	Size of patches	Duration	Other skin lesions	Hair pull test	Hair density	Hair follicle	White dots	Pigmentation	Empty follicles	Dystrophic hair	Follicular plugs	Follicular casts	Histopathology
1	40	F	House wife	S	Patchy	2	3*2 cms	1 year	Nil	-	Reduced	Reduced	-	Ivory white areas	+	+	-	-	PFI
2	35	F	House wife	S	Patchy	3	2*1 to 4*4 cms	2 year	Nil	-	Reduced	Reduced	-	Mild perifollicular brown pigmentation	-	-	-	-	PFI

FRONTAL FIBROSING ALOPECIA

Sl. No.	Age	Sex	Occupation	Clinical type	Type of hair loss	Duration	Other skin lesions	Hair pull test	Hair density	Hair follicle	White dots	Pigmentation	Empty follicles	Follicles with single hair	Dystrophic hair	Follicular plugs	Follicular casts	Histopathology
1	26	F	House wife	S	Hair loss along frontal hair line & sidelocks	1 year	Nil	Negative	Reduced	Reduced	-	Ivory white areas	-	Many	-	-	+	PFI, Infiltrate around arrector pilorus

FOLLICULITIS DECALVANS

Sl No.	Age	Sex	Occupation	Clinical type	Type of alopecia	Duration	Hair pull test	hair density	Hair follicles	Follicular pustules	Crusting	Histopathology
1	23	F	Student	Scarring	Pustules in frontal scalp	3 months	Negative	Reduced	Reduced	Present (Different stages of development)	Perifollicular haemorrhagic	Not conclusive
2	34	M	Office desk	Scarring	Patchy with follicular pustules	9 months	Negative	Reduced	Reduced	Present (Different stages of development)	Perifollicular haemorrhagic	Perifollicular lymphocytic infitrate with perifollicular fibrosis

APLASIA CUTIS

Sl. No	Age	Sex	Occupation	Clinical type	Type of alopecia	Number of patches	Size of patch	Duration	Hair density	Hair follicle	Stellate scars
1	1 year	M	-	Scarring	patchy	1	3*2 cm	Since birth	Absent over patch	Absent over patch	Present all over scalp

NEVUS SEBACEOUS

Sl. no.	Age	Sex	Occupation	Clinical type	Size	Duration	Hair density	Hair follicles	Lipid globules	Histopathology
1	12 y	M	Student	Verrucous plaque with no hair follicles	2*1 cm	Since birth	Absent over lesion	Absent over lesion	Present	Multiple sebaceous glands in the dermis
2	6y	M	-	Verrucous plaque with no hair follicles	3*1.5 cm	Since birth	Absent over lesion	Absent over lesion	Present	Multiple sebaceous glands in the dermis

MONILETHRIX

Sl. No.	Age	Sex	Occupation	ClinicalType	Duration	Associations	Hair density	Hair follicles	Empty follicles	Hair shafts	Other body sites
1	2 months	F	-	? Congenital atrichia- scalp, eyebrow	since birtth	Hypoplastic thumb ASD	Reduced	Present	Present	Nodal dialations with breaks at internodal constrictions	Eyebrows- similar findings

ABBREVIATIONS

- M- Male
- F- Female
- NS- Non-scarring
- FTR- Fronto-temporal recession
- TF- Thinning over frontal region
- TFV- Thinning over frontal region and vertex
- TFVT- Thinning over frontal, vertex and temporal region
- FP- Follicular plugging
- IFEA- Interfollicular epidermal atrophy
- BCDORS- Basal cell degeneration of outer root sheath
- PFI- Perifollicular infiltrate
- PI- Pigment incontinence
- CB- Civatte bodies

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI –3

EC RegNo:ECR/270/Inst./TN/2013 Telephone No : 04425305301
Fax : 044 25363970
Date:10.09.2013

CERTIFICATE OF APPROVAL

To
Dr.Vivek Shah,
2nd year MD(DVL)Post Graduate,
Madras Medical College, Chennai-3.

Dear Dr.Vivek Shah,

The institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Clinical study of dermoscopic findings in non-cicatricial and alopecias" No.13092013.

The following members of Ethics Committee were present in the meeting held on 10.09.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. Kalai Selvi -- Member
   Prof of Pharmacology, MMC, Ch-3
6. Prof. Siva Subramanian, -- Member
   Director, Instt. of Internal Medicine, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

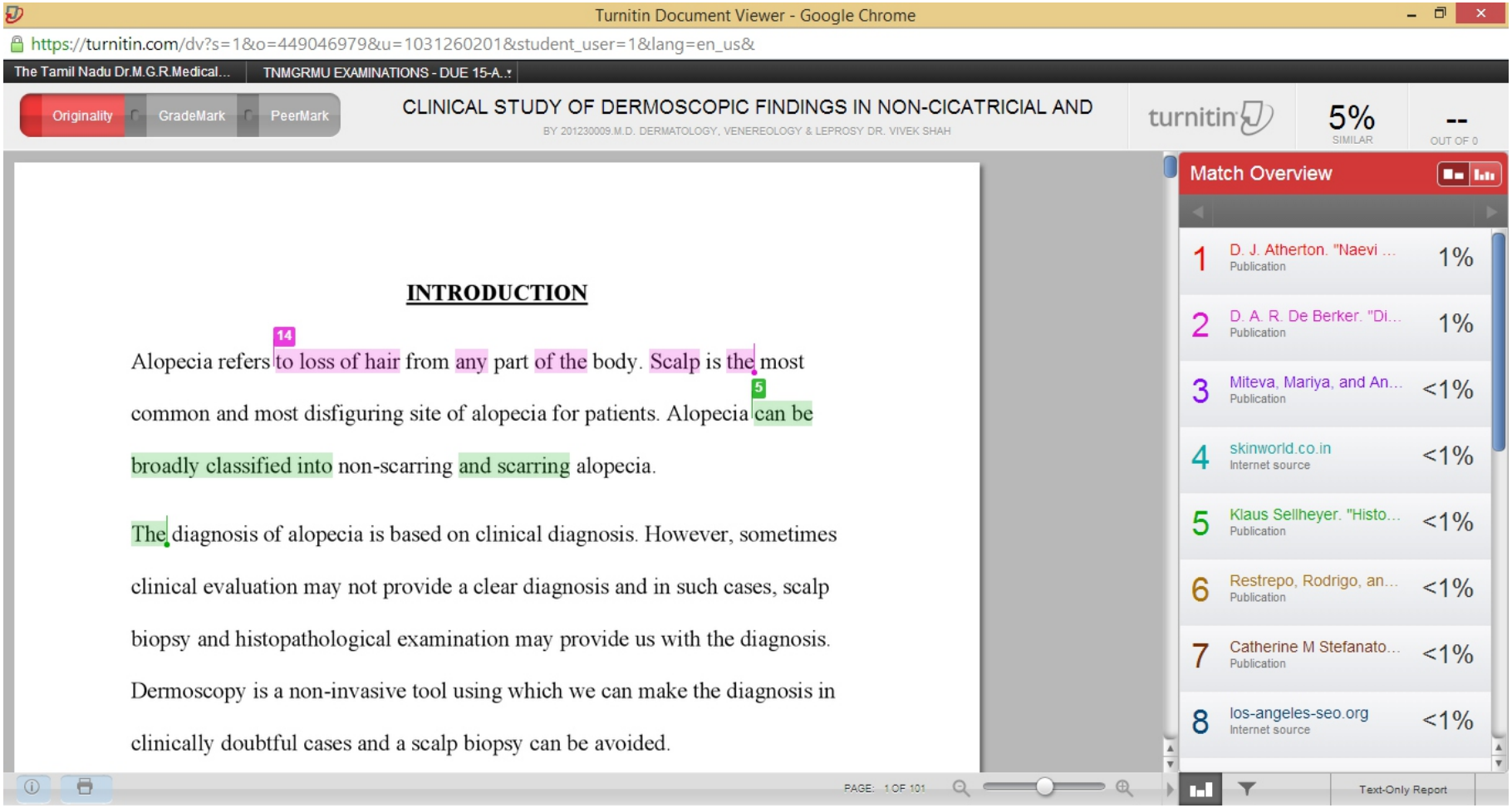
Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003


Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?s=1&o=449046979&u=1031260201&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 15-A..
Originality
GradeMark
PeerMark
CLINICAL STUDY OF DERMOSCOPIC FINDINGS IN NON-CICATRICIAL AND
BY 201230009.M.D. DERMATOLOGY, VENEREOLOGY & LEPROSY DR. VIVEK SHAH
turnitin
5%
SIMILAR
--
OUT OF 0
INTRODUCTION
Alopecia refers to loss of hair from any part of the body. Scalp is the most common and most disfiguring site of alopecia for patients. Alopecia can be broadly classified into non-scarring and scarring alopecia.
The diagnosis of alopecia is based on clinical diagnosis. However, sometimes clinical evaluation may not provide a clear diagnosis and in such cases, scalp biopsy and histopathological examination may provide us with the diagnosis. Dermoscopy is a non-invasive tool using which we can make the diagnosis in clinically doubtful cases and a scalp biopsy can be avoided.
PAGE: 1 OF 101
Match Overview
1 D. J. Atherton. "Naevi ... Publication 1%
2 D. A. R. De Berker. "Di... Publication 1%
3 Miteva, Mariya, and An... Publication <1%
4 skinworld.co.in Internet source <1%
5 Klaus Sellheyer. "Histo... Publication <1%
6 Restrepo, Rodrigo, an... Publication <1%
7 Catherine M Stefanato... Publication <1%
8 los-angeles-seo.org Internet source <1%
Text-Only Report